LANE MEDICAL LIBRARY STANFORD
2 45 0058 7976

LANE

MEDICAL LIBRARY

LEVI COOPER LANE FUND

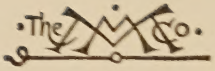
Digitized by the Internet Archive
in 2025

https://archive.org/details/clinicalaspectso0000jaee

MACMILLAN MEDICAL MONOGRAPHS

GEORGE R. MINOT, M.D., S.D., EDITORIAL ADVISOR

THE CLINICAL ASPECTS OF VENOUS PRESSURE



THE MACMILLAN COMPANY
NEW YORK · BOSTON · CHICAGO · DALLAS
ATLANTA · SAN FRANCISCO

MACMILLAN & CO., LIMITED
LONDON · BOMBAY · CALCUTTA
MELBOURNE

THE MACMILLAN COMPANY
OF CANADA, LIMITED
TORONTO

THE CLINICAL ASPECTS OF VENOUS PRESSURE

BY

J. A. E. EYSTER, B.SC., M.D.

PROFESSOR OF PHYSIOLOGY, UNIVERSITY OF WISCONSIN,
ASSOCIATE PHYSICIAN, WISCONSIN GENERAL HOSPITAL,
MADISON, WISCONSIN.

NEW YORK
THE MACMILLAN COMPANY
1929

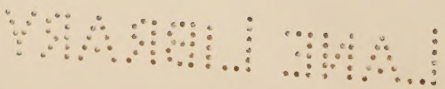
LANE LIBRARY

Copyright, 1929,
By THE MACMILLAN COMPANY.

Set up and printed.
Published June, 1929.

All rights reserved, including the right of reproduction
in whole or in part in any form.

SET UP BY BROWN BROTHERS LINOTYPERS
PRINTED IN THE UNITED STATES OF AMERICA
BY THE CORNWALL PRESS



7
29

INTRODUCTION

WITHIN the past two decades important advances have been made in the knowledge and application of arterial blood pressure determinations in clinical medicine. The significance of venous pressure, on the other hand, has in comparison been greatly neglected. While the latter appears not to have the same extent of widespread interest as the former in a number of more or less diverse clinical states, recent work has tended to emphasize its paramount importance in present or impending cardiac decompensation from any cause, or when there is reason to anticipate the possibility of this event during the course of several diseases. Venous pressure is the most direct indication that can be obtained clinically of the extent to which the heart is moving its load of blood from the venous to the arterial side of the circulatory system, and it exhibits changes which precede the development of other signs and symptoms when the heart fails to do this to the normal degree. By far the greater part of the clinical situation in cardiac failure is due directly to venous engorgement, exhibited as passive congestion in various organs, and as exudates from serous membranes. As such engorgement occurs, the venous

71167

pressure rises, to fall again as the engorgement is reduced or disappears.

Venous pressure may therefore be said to represent not only the primary factor that underlies the symptoms and functional pathology of cardiac failure, but to be also responsible in large part for the physical signs accompanying it: edema, congestion, cardiac dilatation, and reduced urine secretion. As the principal underlying factor in these conditions, and as the principal index of cardiac behavior, it is the most reliable and important single factor to know and to follow accurately when this clinical state develops, or when it is impending.

Determination of venous pressure by the indirect method of vein compression is accurate, and can be readily and quickly carried out. It requires no more experience in order to secure confidence and accuracy than arterial pressure determinations. Although this method has been available and its importance more or less evident for a number of years, it has had by no means widespread clinical use in this country or abroad. It is hoped that the present book, written primarily for clinicians, and to stress the clinical application of venous pressure, will stimulate an interest which can only make for a better recognition, understanding, and treatment of cardiac decompensation. Since an understanding of normal conditions must precede the interpretation of abnormal states, a brief review and presentation of the present conception of the cause and sig-

nificance of venous pressure is given, followed by a discussion of the altered state that exists in cardiac decompensation. The conception of this condition which is presented has come in many of its details only recently through a more thorough understanding of cardiodynamics, especially in its relation to venous pressure. Following this, the method and its clinical applications are discussed.

For those who may desire more complete references to the original literature than are given, the bibliographies occurring in the principal papers quoted cover quite fully the literature up to the present time. The numbers appearing in parenthesis in the body of the text refer to this literature as listed in the bibliography at the end of the book.

CONTENTS

		PAGE
	INTRODUCTION	v
CHAPTER		
I.	THE MECHANICS OF VENOUS PRESSURE IN THE NORMAL AND ITS RELATION TO CARDIAC ACTIVITY	1
II.	CARDIAC DECOMPENSATION	14
III.	METHODS	19
IV.	VENOUS PRESSURE RANGE IN THE NORMAL INDIVIDUAL AND THE INFLUENCE OF VARIOUS FACTORS	32
V.	VENOUS PRESSURE IN CARDIAC DECOMPENSATION	40
	Relation to Clinical Course	42
	Relation of Venous Pressure to the Water Balance	47
	Relation of Venous Pressure to Cardiac Dilation in Decompensation	57
	The Capillary Pressure and Flow in Cardiac Decompensation and Its Relation to Venous Pressure in This and in Other Conditions	61
	The Effect of Therapy on Venous Pressure in Cardiac Decompensation	65
	Cardiac Failure in Lobar Pneumonia	79
VI.	VENOUS PRESSURE IN CONDITIONS OTHER THAN CARDIAC FAILURE	82
	Chronic Heart Disease	82
	Paroxysmal Tachycardia and Fibrillation	82

CHAPTER PAGE

Miscellaneous Conditions Involving the Cardiovascular System 83
Periphreal Vasodilation 83
Arterial Hypertension 84
Pulmonary Conditions Other Than Pneumonia 84
Local Venous Obstruction 87
Miscellaneous Disease 89
Venous Pressure in General Anesthesia 89

VII. Clinical Cases 91
Endocarditic Group 91
Myocardia Group 92

VIII. Summary 115

Bibliography 123

LIST OF ILLUSTRATIONS

FIG.		PAGE
1.	Approximate blood pressures in different parts of the vascular system in normal man at rest in the recumbent position	2
2.	Apparatus for measuring venous pressure by the indirect method	23
3.	Chart showing venous pressure, fluid intake and urine output in Case I	95
4.	Chart showing venous pressure, fluid intake and urine output in Case II	99
5.	Chart showing venous pressure, fluid intake and urine output in Case III	101
6.	Chart showing venous pressure, fluid intake and urine output in Case IV	105
7.	Chart showing venous pressure, fluid intake and urine output in Case V	109

THE CLINICAL ASPECTS OF VENOUS PRESSURE

THE CLINICAL ASPECTS OF VENOUS PRESSURE

CHAPTER I

The Mechanics of Venous Pressure in the Normal Individual and Its Relation to Cardiac Activity.

The physical conditions existing in the systemic circulation determine a progressive fall of blood pressure through the arterioles, capillaries, venules, and veins. The fall of pressure is due to a conversion of the original head of pressure (resistance head), or driving force, into heat as a result of friction. The friction results from the resistance offered to the passage of blood through the vascular channels, and is proportional to the wall surface area in any part of the system. The resistance, and hence the dissipation of the energy of the head of pressure occurs most rapidly in the arterioles, capillaries, and venules because of their large wall-surface area, so that by the time the larger veins are reached a comparatively low pressure is encountered. The accompanying chart (Fig. 1) gives the approximate average blood pressures found in man at bed rest. Arterial pressure shows considerable variations as a

result of normal influences, such as body position and activity, and the same is true of venous pressure. Under conditions of rest in the recumbent position, both arterial and venous pressures, and

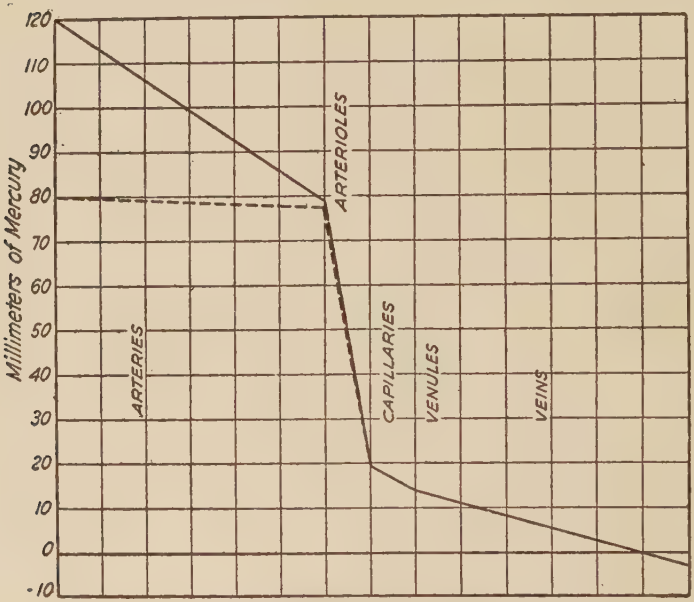


Fig. 1.—Approximate pressures in different parts of the vascular system in man.

especially the latter, tend to assume a more constant figure. Arteriole, capillary, and venule pressure have not been studied so thoroughly, but would seem to vary somewhat more than venous, but less than arterial pressure. Compared with atmospheric pressure the venous pressure at the end of the sys-

tem (venæ cavæ) is at, or below, zero, but is definitely positive as compared with the pressure present in the thorax (intrathoracic pressure). The blood thus normally returns to the heart with definite, although small, pressure remaining. The pressure in the arteries is ordinarily expressed in millimeters of mercury, and this measure is also applied to arterioles, capillaries, and venules. By the time the large veins are reached, the pressure has fallen so low that a more convenient measure is millimeters of water. Mercury is 13.5 times as heavy as water, and one method of measurement can be converted into the other by the use of this factor. Measurements of the venous pressure in the larger veins of the body surface, such as those on the dorsum of the hand or foot, show considerable variations under ordinary conditions, determined largely by physical activity and the position of the vein in reference to heart level. The latter variations are the result of the effect of gravity. While this effect influences all parts of the circulation, its result is relatively much greater upon venous pressure, because of the low residuum from the original head of pressure, derived from the heart beat, which is present in these vessels. Under ordinary conditions the pressure in any superficial vein of the trunk or extremities is the result of two factors: the residuum of the head of pressure created by the heart beat, and the influence of gravity. The latter is determined by the height of the

column of blood that is present between the point measured and the entrance into the right auricle.

In the veins of pendant parts, such as the hands or feet, the venous pressure does not ordinarily rise as high as these two factors would indicate, nor even as high as gravity alone would determine, because of the influence of contracting skeletal muscles and the disposition of the venous valves (1). Each time a group of muscles contracts, mechanical pressure is exerted upon the contained and surrounding veins, and blood tends to be forced from them. Because of the interposition of the venous valves, this blood flows toward the heart. In a pendant, inactive limb the venous pressure gradually rises to a high point, sufficient, probably, to stop all circulation for a time in the part concerned. The influence of gravity amounts to approximately 10 mms. of water or about 0.74 mm. of mercury for each vertical centimeter. For determination of what may be designated as clinical venous pressure in any vein, the subject should be in a condition of physical rest, in the recumbent position, and with the vein at approximately the level of the entrance into the right auricle, or the measurement corrected and referred to this position. Under these conditions it has been shown that in normal man the venous pressure in one of the larger superficial veins is quite constant in the same individual from time to time. It shows only a narrow range of readings in different subjects, and age exerts little, if any, influence. The usual

reading obtained is from 40 to 60 mms. of water, and rarely, if ever, exceeds 110 mms. (2).

Modern physiological work has established two factors which appear to be of particular importance in stabilizing venous pressure under normal conditions within relatively narrow limits: the variable capacity of the peripheral vascular bed (arterioles, capillaries, and venules); and changes in the amount of blood pumped by the heart. In regard to the former, the conception of a variable tonus or calibre, ascribed originally only to the arterioles through the vasomotor system, has been extended to include also the capillaries and venules (3) (4) (5). It has been shown that the blood flow through many capillaries ceases when a part is at rest (6). For example, if one observes, under the microscope, the capillary loops at the base of the finger nail under resting conditions, only about one-half or two-thirds will show corpuscles streaming through them. In the remainder the blood is stagnant. When this part becomes active, the capillaries become patent, and the volume of peripheral blood and blood flow increases. It has been shown both in the experimental animal (7) (8) and in man (9), that the blood volume may be altered considerably, as much as ten per cent of its total, without affecting more than momentarily arterial or venous pressure, or the blood flowing into the heart. This varying blood volume has been accommodated by variation in the peripheral vascular bed. The second factor, variations in the extent of

heart activity, by means of which the amount of blood it pumps is varied, and determined to a large extent at least by the pressure of the blood returning to it through the venae cavae is an important one, not only from the standpoint of the physiological conception, but also because it appears to be the most important mechanism that is altered in heart failure or decompensation. It has been established that the normal heart tends to put out on the arterial side as much blood as comes to it from the venous side, even though the latter may vary through rather wide limits, and that it thus prevents engorgement or depletion of the blood in the larger veins, and marked alterations in pressure. The situation would appear to be somewhat analogous to that existing in the respiratory mechanism. In the latter, the oxygen and carbon dioxide content of the blood is maintained at an approximate level by variable activity of the respiratory center, leading to variation in the depth and rate of breathing, even in the presence of wide variations in the amount of oxygen consumed, and carbon dioxide produced, by the body. A variable venous return to the heart is conditioned largely by variations in muscular activity. When a group of muscles enters into activity, the venous blood contained in them is in large part forced out by compression of the veins, and the complement of blood that they receive from the arteries and pass on to the veins is greatly increased, due to the local enlargement of the vascular bed. This

results in an increased venous return to the heart, and a rise of pressure in the larger veins. The increased blood return to the heart quickly augments cardiac activity, which tends to restore venous pressure to its previous level. The augmentation of cardiac activity, leading to increased output of blood from the heart, is due in part to increased rate of the heart beat (10) (11), and in part to increased filling and increased contraction (stroke volume) in each beat (8). The rate at which ejection of blood occurs during the contraction phase appears also to be increased (12).

The heart acts merely as a force pump, and does not differ in its mechanics from the usual mechanism of this type. It consists of chambers, the capacities of which are varied, and valves so disposed that flow can occur in one direction only. Blood is received into it through the inlet valve (auriculo-ventricular valve), which is open so long as the pressure within the pumping chamber (ventricle) is below that in the receiving chamber (auricle). When the ventricular muscle contracts, and begins to exert pressure on the enclosed blood, the auriculo-ventricular valve closes because of a higher pressure in the ventricle than in the auricle, and as this force continues to be exerted, the aortic valves are forced open, and blood begins to flow out into the arterial system. This outflow continues so long as the contraction of the ventricular muscle is sufficiently powerful to maintain a higher pressure in the ventricles than in

the aorta. As the contraction phase begins to pass off, the period of outflow ends coincidentally with the closure of the aortic valves. A short period now elapses in which the ventricular pressure is falling to a point equal to and below the existing auricular pressure. When this point is reached, the auriculoventricular valves open, and the ventricle begins to fill with blood in preparation for the next stroke. It is obvious that the amount of blood that the ventricle expels at each beat (stroke volume) will depend upon two factors: the amount of blood that it receives from the auricle during its period of relaxation, and the extent to which it reduces its volume during the period of contraction. The amount of blood expelled, in other words, is directly proportional to the degree of relaxation and the extent of contraction of the ventricular muscle fibers.

Physiological experimentation has shown that this variable factor, the extent of contraction of the muscle, is mainly, perhaps entirely, determined by the pressure of the venous blood. What is the relation between these two factors? To understand it we must go back to a physiological relationship established a number of years ago and repeatedly confirmed by modern work, and which is concerned with the relation that exists between the extent of contraction or energy released by a skeletal muscle during its contraction, and the load which is placed upon it at the onset of its contraction (initial load). If one isolates the gastrocnemius muscle of a frog and

suspends it so that its contraction can be recorded by a lever, and in such a manner that weights of varying degree can be attached to it, it is found that on stimulation, the energy released (extent of contraction multiplied by the load moved) is, within certain well-defined limits, proportional to the load that is present. A light weight may be associated with quite a large contraction, but little work is done. With a heavier load, the muscle starts to contract at a lower point, due to the stretching of its fibers under the influence of the load; and, while it may not contract to the extent it did formerly, the amount of work done is increased. As the load increment is still further increased, the muscle stretches more and more. With each contraction it does more and more work, until a point is reached where the work curve (product of load times lift) begins to decrease, and finally the load is not moved at all, and the work becomes zero. The result may be stated as a general law: with increasing initial load, a muscle during contraction increases its energy release in the form of mechanical work up to a certain value of load. This upper limit of load represents the upper limit of physiological response of the muscle. With each increment of load the muscle is subjected to increased stretching and increased tension, and it is believed that one or both of these factors is the determining cause of the increased energy release. By many it is believed that the essential factor in muscle contraction is a surface phenomenon

of some sort occurring on the fiber interfaces, and that the result of the increased load is to increase the surface area of the interfaces, and thus lead to an increase in the process, whatever its nature may be, which occurs on these interfaces, and which is responsible for contraction. Others regard the main factor as a matter of tension of the individual fibers, a relation existing between the fibers' own tonus and the pull which is being exerted upon it. Whether one or both of these factors determine the result can only be answered by further work. The essential point at present is the direct relationship that exists between initial load and work response, and the limitation of the muscle when it is stretched beyond its physiological limits by a load that is too heavy to be moved effectively. Recent studies have extended this conception to cardiac muscle, and have shown that the relationship that exists here between initial load and work response follows the same general law, and differs in no essential respect from skeletal muscle (13) (14). The initial load upon the ventricle is the pressure of the blood that is contained within its chamber at the onset of its contraction. This in turn depends upon the extent of its filling, as determined by the supply and pressure of the venous blood returning to it. This load is augmented, just before ventricular contraction, by the systole of the auricle, which adds the last complement of venous blood to the ventricle. The auricular muscle follows the same general law relating load

and work; and the extent of its contraction. The extent of augmentation of initial intraventricular pressure or load hence varies with the venous pressure of the returning blood. With a large flow through the peripheral vessels and a large return to the heart, the venous pressure rises. More blood, and blood under a higher pressure, flows into the auricle and ventricle during diastole. Both are stretched and put under a higher tension than before. The contraction of the auricle occurring toward the end of diastole is stronger, and adds still further to the amount and pressure of the blood in the ventricle. The ventricle is still further stretched, and put under a still greater tension. It starts contracting with a larger complement of blood, and moves a greater quantity of it into the aorta before its contraction is completed. Estimates in man and animals, made by somewhat indirect methods, show that under normal conditions, associated with the degree of physical exertion, the stroke volume may increase from three to six times. Assuming a doubling of the rate under conditions of considerable exertion, it is obvious that the capacity of the normal heart to provide for increased demands of blood supply is large.

There is thus ordinarily a large reserve factor, more than sufficient to meet the usual demands. Indeed, recent work tends to show that even the most extreme demands in the normal heart do not exceed its reserve capacity. The most extensive venous return that can occur in the intact animal appears

to exert a load which is still within the physiological limits of response of the normal cardiac muscle. The final limiting factor in the normal circulation, under abnormal loads produced experimentally, and of a magnitude which is probably never closely approached under the usual environment, appears to be not the heart but the peripherial vascular system (15).

The two mechanisms that have been considered, the variable vascular bed and the adaptation of cardiac activity to the venous return, with the addition of variations in peripheral blood flow, are the important mechanisms that determine venous pressure under normal conditions. The variable vascular bed may be regarded in part as a buffer protecting the heart against sudden and extreme variations in venous load. The relation of cardiac activity to venous pressure represents an adaptable mechanism, by means of which the output from the heart into the arteries and capillaries is largely determined by the magnitude of the return flow of blood from these vessels. These two mechanisms assure that, under normal conditions, heart load will not exceed the range of physiological response of the cardiac muscle, and that the heart will pump into the arterial side of the system the blood that comes into it from the veins, without more than a temporary rise of venous pressure.

That cardiac activity, represented by the response of the heart to the venous pressure load, rather than

peripheral conditions in the circulation, is the principal factor in determining venous pressure, is evident from a number of observations. Warming or cooling the skin surface locally, while altering the size and degree of filling of the larger superficial veins, does not appreciably alter the pressure of the blood within them (16). Even in the extreme peripheral constriction encountered in Reynaud's disease, venous pressure is unaffected (17). Peripheral alteration of tonus in the arterioles and venules affects the pressure in these vessels and the amount of blood flowing through them into the veins, but does not affect venous pressure to any noteworthy extent. Venous pressure, while representing in part the terminal pressure exerted by the heart through the peripherial vascular system, is so closely connected with cardiac activity through its influence directly upon the heart, that this must be regarded as probably the most important factor controlling it, and tends to stabilize it, at least under conditions of rest, within a relatively narrow range.

CHAPTER II

Cardiac Decompensation

Cardiac failure or decompensation occurs when the initial load on the ventricle, represented by the venous pressure, exceeds the upper limit of physiologic response of the cardiac muscle. The state is reached in which the load is too great to be moved effectively. The muscle is stretched beyond its physiological limit, and the contractions are weak and inefficient. This situation may be conceived of as resulting from one of two, or a combination of two, factors; an abnormal increase of venous load, or a narrowed range of response on the part of the cardiac muscle, within which it normally acts by increasing its energy of contraction to increased load. As we have seen, it is most probable that the load can never become too great, except under experimental conditions, to exceed the upper range of response of the normal cardiac muscle, so that we must assume a narrowed range of response as one of the factors in cardiac decompensation. This narrowed range of response is primarily a result of disease of the muscle, and decompensation occurs when this range is so reduced that the normal variations of venous pressure, or perhaps the somewhat

higher venous pressure resulting from unusual exertion, exceeds its upper limit. While it has been shown that total blood volume is not ordinarily increased in cardiac decompensation (18), the attending vasoconstriction, by means of which a normal or increased arterial pressure is maintained in the presence of a diminished cardiac output (19) (19a), and the failure of the heart to move the usual amount of blood in the veins into the arteries, results in a stagnation of the blood in the veins, and a proportional increase in the blood volume in this part of the circulation. Venous pressures are high throughout, and edema, and engorgement due to blood stasis, supervenes. It is probable that the liver, with its double set of peripheral vessels, is particularly important as a variable blood reservoir normally feeding into the venae cavae (21), and this is one of the organs most affected when venous engorgements result from cardiac failure. Recent work has also emphasized the function of the spleen in the same relation (22), and here again massive enlargement may occur from the abnormal venous engorgement. It has been known for many years that obstruction to the venous flow from the kidney results in a decrease in urine secretion. This has become, indeed, a classical experiment in the history of the physiology of the kidney, in that it was employed by Haidenhein to refute Ludwig's conception of the relation of kidney secretion to filtration occurring from the blood stream in the Bowman capsule. Ludwig, in turn, showed that the decreased urine

under these circumstances could well result from the encroachment upon, and actual occlusion of, the tubule by the engorged interlobular veins. That there is a direct relationship between venous pressure and urine formation in cardiac decompensation has been well established. With a rising venous pressure in this condition, the amount of urine decreases, to increase again as the venous pressure falls, and careful observation shows that the venous pressure changes precede the changes in kidney function (23). The pulmonary edema is probably likewise to be ascribed to the venous engorgement and high capillary pressure in this vascular circuit. It has been shown that the reduction of the vital capacity of the lungs in decompensation bears a direct relation to the degree of this involvement. Pleural and peritoneal transudates accompanying decomposition, represent results from increased filtration due to the high capillary and venous pressures present.

It has been shown experimentally that the amount of oxygen consumed by heart muscle per unit of work increases with the initial load placed upon the muscle (24), and if this load exceeds the normal range of adequate response, the oxygen consumption rises to an abnormal height (25). The heart in decompensation is thus inefficient as regards oxygen utilization, requiring an excessive supply. This occurs, of course, in the presence of a reduced supply. The slowing of the pulmonary circulation leads to deficient blood aeration, while the returning systemic

venous blood has had a higher percentage of its total oxygen removed than under normal conditions. The coronary blood flow is also reduced as a result of the dilatation of the muscle (26), and the rapid and weak contractions (27) (28). Lack of sufficient oxygen supply, or anoxemia of the heart muscle, is thus an important factor in decompensation, and results in a further reduction of cardiac efficiency. This leads in turn to a still greater increase in venous stagnation and increase of the venous load. The "vicious cycle" of cardiac incompetence thus becomes established. The therapeutic indications for the reduction of muscular and other activities to as near basal conditions as possible is obvious from the standpoint of the oxygen requirements of the tissues, and in lowering the demand for peripheral blood flow, and consequently the venous return to the heart. The value of venesection in reducing the proportionately high venous blood volume and abnormal initial load on the right ventricle is equally clear. It is probable that digitalis acts in large part by reducing the heart rate, and in bringing about through this a more efficient utilization of oxygen by the cardiac muscle. It has been shown experimentally that a high rate of contraction leads to rapid heart failure in asphyxial conditions (29). The improved oxygen utilization and the increased coronary flow resulting from stronger and slower contractions, along with the lowered venous pressure offering less hindrance to the return flow from the

coronary veins, are important factors in reducing the anoxemia of the heart muscle, as compensation begins to be restored.

Cardiac compensation is regained, when by improvement in the function of heart muscle and by reduction of the venous engorgement by rest, venesection, or other means, the muscle begins to respond to the increased load by increased work. As the muscle becomes more efficient in its response, the venous load diminishes, and cardiac output and venous pressure return to within their normal ranges. The load is again within the range of adequate response on the part of the cardiac muscle.

That cardiac dilatation occurs during periods of decompensation, to be again reduced or disappear as compensation is restored, has been shown by the usual methods of physical examination, and confirmed by the more accurate methods of x-ray determination of heart size (2). It has also been shown that there is a direct relationship between the extent of cardiac dilatation and the rise of venous pressure. Venous pressure is thus an indication of the presence and extent of cardiac dilatation in decompensation. Because of its ease and accuracy of determination, it is to be regarded as more reliable than are inferences derived from cardiac percussion, or other means employed to determine this condition from the heart itself.

CHAPTER III

Methods

Two general methods, which may be designated as the direct and indirect methods, have been used for the determination of venous pressure in man. In the former, a rather large trocar is introduced directly into a superficial vein, usually the median vein on the forearm, and connected by a rubber tube filled with physiological saline solution to a water manometer. If the vein is brought to the level of the right auricle, the reading of the manometer should indicate the venous pressure. The indirect method depends upon the principle that if outside pressure is exerted upon a superficial vein, the vein will be either patent or collapsed, depending upon whether the outside pressure is lower or higher than the pressure of the blood within the vein. Theoretically, the actual venous pressure is between the outside pressure required to collapse the vein, and the outside pressure just sufficient to allow the vein to fill. Practically, it is found that these two points are so close together that either may be used and sufficient accuracy ensured. Since the principal value of the measurements is comparative rather than absolute, and since the least pressure required to produce col-

lapse of the vein is more readily determined, this, rather than the pressure allowing patency, is usually employed. The tendency of the vein to remain patent independent of any pressure within it, or, as may be expressed in another way, the pressure required to collapse a vein with zero pressure within, is negligible, because of the thin walls and non-rigid character of the vessel.

The first means employed in the application of the indirect method, which was the first effort to measure venous pressure in man [Frey (30)], was by the use of a bar resting on the skin, movable in a vertical plane, and to which weights were added until collapse of the vein occurred. Knowing the surface area exposed, and the amount of weight, the pressure could be calculated. Shortly after this [v. Basch (31)], the principle of using an air chamber over the vein and applied to the skin surface, through which the vein could be observed, and into which air could be forced under pressure, was proposed. It is this principle which is employed in the modern type of apparatus used for indirect measurement. The air pressure in the chamber over the vein is read on a water manometer connected with it, and the amount of pressure necessary to collapse the vein is determined by the height to which the manometer column is forced, as expressed in millimeters or centimeters of water. In 1908 an apparatus using this principle was described by the writer in collaboration with Dr. D. R. Hooker (32). The

air chamber consisted of a box made of aluminum about two inches square, the top and one side of which was of glass, and the bottom covered by a replacable dental dam membrane. The rubber dam was provided with a hole in the middle, and was coated with glycerin before application to the skin over a vein, in order to secure good adhesion to the surface, and to render the chamber air tight. The box was connected by a rubber tube through a side opening to a U manometer provided with colored water and a scale reading in half centimeters. A side tube from this connected to a rubber bulb which could be squeezed within a clamp, air pressure thus being produced in the chamber and on the column of water. The vein was observed through the glass top and hole in the middle of the rubber dam. A prominent vein on the back of the hand or forearm was selected and brought to approximate heart level, the air chamber applied, and the vein observed as the pressure was increased. At the moment of collapse of the vein the water manometer was read. Hooker (33) subsequently described a modification in which the manometer was made smaller and more easily portable, and the original air chamber was replaced by a glass capsule, which was cemented to the surface of the skin by celloidin. The apparatus originally used by Hooker and Eyster has been recently redesigned by the writer with the assistance of Mr. J. S. Hipple, Mechanician of the Medical School, University of Wisconsin.

The principal objects kept in view were the ease and rapidity of replacement of parts, portability, and protection against loss of fluid from the manometer. The original square box, which was used as an air chamber over the vein, has been replaced by a round one, which is more easily manufactured, and allows rapid replacement of the rubber dam covering when necessary.

This chamber, shown in actual size in the detail drawing (Fig. 2), is 1¾ inches outside diameter, and consists of two parts. The inner part **1** is a metal ring, curved on its lower surface to fit the contour of the back of the hand or forearm, and provided with an outlet tube **2** at one point. This ring is closed above by a glass disc **3**, held in place by a flange below, and a screw ring **4** above. The outer part **5** is another ring, the inside diameter of which is slightly larger than the outside diameter of the first ring, so that it fits somewhat loosely over it. It has the same depth as the other ring, and is notched at one place to fit over the outlet tube **2**. It is also curved along its lower margin in the same manner. Rubber membrane of the thickness known as "dental dam" is placed over the lower margin of the inner ring **6** and tied with thread. There is a groove around the surface of the ring to prevent the thread from slipping. The outside ring is now slipped on, and the free margins of the dam cut away. An opening about half an inch square is cut out of the middle of the rubber dam **7**.

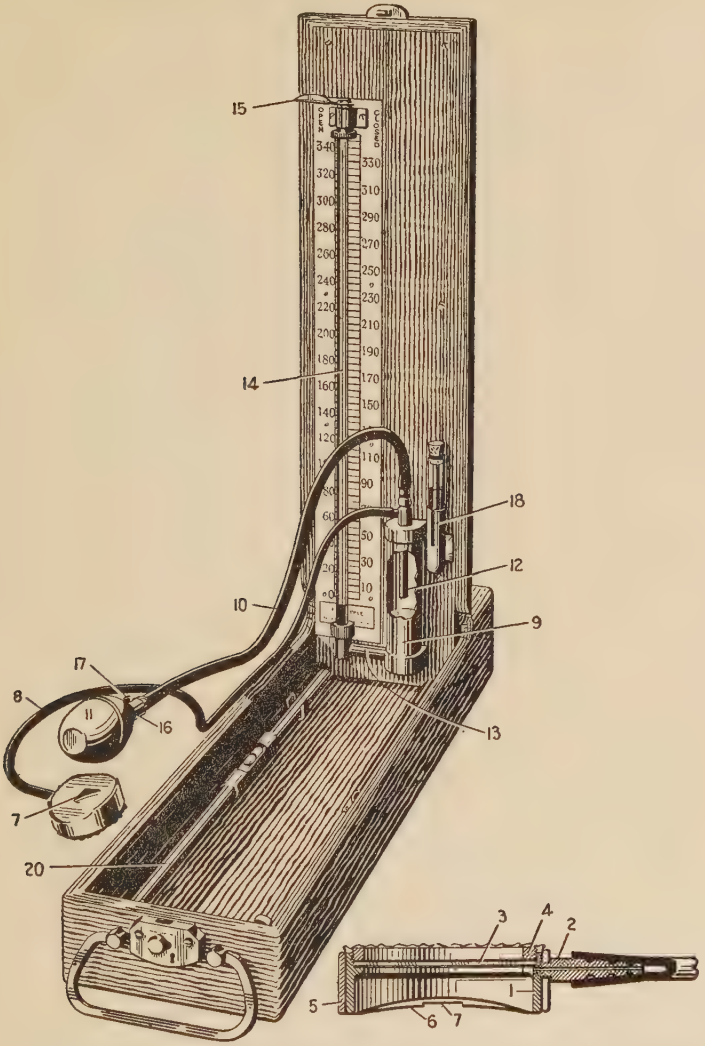


FIG. 2.—Apparatus for the determination of venous blood pressure in man by the indirect method.

Rubber tubing **8** about 20 inches long connects the outlet from the air chamber to a metal cylinder **9**, which forms one arm of the manometer. This cylinder is 3⅞ inches long, and ⅞ of an inch in diameter. From the upper end, a tube projects, which is connected by rubber tubing **10** to the pressure bulb **11**. This tube also projects into the metal cylinder **12**, to prevent loss of fluid when the manometer is not in a vertical position. It also gives off a lateral tube which is connected with the air chamber as described above. From the lower end of the cylinder, a lateral tube **13** passes to connect with a metal mounting forming the base of the glass manometer tube **14**. This tube is 5/6 of an inch in outside diameter, 13¼ inches long, and is held at the top by a metal collar and mounting. The upper opening to the outside air may be closed to prevent loss of fluid when the manometer is not in use. This opening is controlled by a short projecting handle **15**, which is so placed that when it is in the open position (turned to the left) the cover of the box cannot be closed. When the manometer is in use, it should be, of course, in the open position.

The pressure bulb **11** is of the usual rubber type, 1½ inches in diameter, and 2¾ inches long. It is fitted to a metal tube **16** 2½ inches long, adapted to receive the bulb at one end, and to make connection with rubber tubing at the other end leading to the metal cylinder as described above. On this metal tube there is a short side projection **17** pro-

vided with a flat surface, and a small outlet hole in the middle of this surface. The hole is kept covered by the thumb of the right hand when increasing the pressure in making a venous pressure determination. Opening the hole by raising the thumb allows air to escape and the pressure to fall.

Back of the manometer column there is a scale on celluloid, graduated to show readings of water pressure for each 5 millimeters. The divisions are actually 4.465 millimeters apart, to make allowance for the change in the short arm of the manometer. The scale provides for readings up to 340 millimeters. The manometer is supplied with water colored with eosin. The metal parts of the manometer are nickel-plated inside and out, to prevent rusting.

The manometer is mounted on the inside surface of the case cover. This is hinged 2½ inches from the end of the box and is held upright by a spring when the apparatus is in use. The case is also provided with metal clips to hold the air chamber when not in use. A bottle of glycerin in a metal mounting **18**, and provided with a stopper and camel's hair brush, is attached to the cover at one side of the manometer.

The level **20** consists of a nickel-plated rod 17 inches long with a glass spirit level mounted in its center. It is bluntly pointed at each end. When not in use, it is held in the box by metal clips, as shown in the drawing.

The case is provided with a snap lock and carrying

handle at one end. It measures 18 x 4¼ x 2¼ inches. The complete apparatus weighs 4½ pounds.

The patient should be at rest in the recumbent or reclining position for at least 15 minutes before the determination of venous pressure is made with this apparatus. A fairly prominent vein on the hand or forearm is selected, and brought to approximate heart level by the use of the spirit level. In the recumbent position, this is taken as one-third the distance from the anterior surface of the thorax to the dorsal surface at the level of the fourth intercostal space. In the semi-recumbent position, the vein is brought to a level with a line joining this space in mid-axilla with the tip of the ensiform process. A light coating of glycerin is now applied to the rubber dam covering the air chamber, and this is placed over the vein and held lightly in contact with the skin by the left hand of the operator. The vein is seen through the glass top of the chamber, and the square hole in the middle of the rubber dam covering. The rubber bulb held in the right hand is now squeezed sharply several times to force out the dam in good contact with the skin, to which it adheres because of its glycerin coating. In occasional subjects it may be desirable to shave the skin surface. The small air-escape hole should be covered by the thumb during this process and the actual determination of pressure, but opened to allow escape of air between different determinations. When good contact has been secured, the air pressure is raised by squeezing

the bulb slowly until the vein is seen to collapse. Cross lighting with moderate illumination is desirable to bring out the vein clearly. After a little experience, the end point of vein collapse is sharp, and easily determined. Reading of the water manometer is made at this point. A number of determinations should be made, until several agree within one centimeter.

Certain modifications of the indirect method have been used. The simplest of these, proposed by Gaertner (24), requires no apparatus, and consists in determining the level as compared with heart level, at which a superficial vein of the hand is seen to go from a state of filling to one of collapse. The hand is first lowered until the vein is full, and then raised slowly. Collapse of the vein occurs at the level where the negative hydrostatic pressure equals the positive venous pressure. If this is compared with the estimated level of the right auricle, one can express the venous pressure in millimeters of water. The vein represents a manometric tube connected with the right auricle. Intermittent or momentary pressure on the vein has been proposed by White (35), in order to avoid the possibility of abnormally high readings due to interruption of the flow in the vein when employing the usual technique of compression. The short periods of pressure are applied through a capsule over a vein, starting with one sufficiently high to produce collapse, then in decrements of 5 mms. of water until the lowest pressure sufficient

to produce momentary collapse of the vein is found. Frank and Reh (36) developed a method using two sphygmomanometer arm bands, one placed around the upper arm, and the other around the forearm. The lower cuff was lightly inflated, and by its connection with a tambour writing on a drum, gave a record of variations in volume of the forearm. The upper arm band was connected with a water manometer, and was inflated slowly until the volume of the forearm began to increase, indicating interruption of the return venous flow. The pressure read at this time was taken as the venous pressure of the superficial veins of the forearm.

The direct method of venous pressure determination by cannulation of a superficial vein was first employed by Moritz and v. Tabora (37) in 1910, and by a number of subsequent workers [Schott (38), Claude, Porox and Rouillard (39), Fuchs (40), La Conte and Yacoel (41), Villeret, St. Girons and Grellity-Bosviel (42), Gazotti (43), Castellotti (44), and Bedford and Wright (45)].

Critical consideration of the two general methods employed, the direct and indirect, leads to the conclusion that for clinical purposes the latter is preferable, since it appears to be at least as accurate as the direct method, and has the great advantage that numerous determinations can be made on a single individual without discomfort or injury. Frequent readings are of great value in impending or actual heart failure, because it is the trend of the venous pressure

as well as its actual height which is important, and this is impossible to determine by direct cannulation of the vein. In nearly all clinical cases where the direct method has been employed, only a single reading has been reported, and while this may be of interest as to the actual height of venous pressure in decompensation, it is of little value in a changing situation or as a control of therapy. While it might be thought on a priori grounds that direct cannulation of a vein with direct connection to a manometer would enable one to make a more accurate determination of the pressure, this does not appear to be the case, because of the difficulties of the method and the considerable chance of erroneous readings. Bedford and Wright (45), and Runge (46) especially, have studied the possible errors in the direct method. The latter observer mentions the use of a trocar with bore so small as to cause capillarity, faulty placing of the trocar in the vein, and occlusion of the connection with the manometer, as the more important causes of failure. He made three readings in every case and discarded those in which there was any considerable discrepancy, those in which a hematoma around the vein occurred, and those in which an expiratory effort with the chest held rigid in inspiration (Valsalva experiment) failed to show a marked rise of venous pressure. Using these criteria, measurements were acceptable on only forty-eight of seventy-five subjects examined. The greatest discrepancy in venous pressure readings recorded in the

literature are those in which the direct method has been used. In the hands of some workers very slight differences between different individuals are recorded (43), while others find the widest variations that have been reported (45) (47). Any one who has used the direct method in the experimental animal is aware of the technical difficulties and many possibilities of error. Several workers have carefully compared the indirect and direct methods in the same animal, and have found that the readings obtained are practically identical.

If the non-instrumental indirect method of Gaertner, described above, is employed, it should be carried out with considerable care, and can only yield readings of approximate value. It is unquestionably of some value, however, where more refined methods are not available. The arm must be raised very slowly to determine the level of collapse of the superficial vein, and several estimates should be made. In using the capsule method, prolonged collapse of the vein, with possible rise of pressure within it, should be avoided; but in the usual technique employed and described above, the periods of collapse are transitory, and cause no significant rise. The method of Frank and Reh, using the two arm bands, shows that even with complete and prolonged collapse, the pressure rises very slowly.

In reference to the relative value of the glass capsule, cemented to the skin over the vein with celloidin, and the chamber employing rubber dam in

which contact is made with the aid of glycerin, each would seem to have its field of usefulness. The former has some advantage when readings are to be made on the same individual at very frequent intervals, every few minutes or less, as in experimental work concerning the effect of exercise, respiratory activity, and other influences. Considerable time and effort, however, is necessary to apply and remove the glass capsule, and for clinical determinations on the same individuals made hours apart, or where a number of patients are under observation, the rubber dam chamber is to be preferred. A disadvantage of the glass capsule, not infrequently encountered, especially in warm weather, is the condensation of water on the inner surface of the capsule, obscuring the view of the vein.

CHAPTER IV

Venous Pressure Range in the Normal Individual and the Influence of Various Factors

The factors which may influence venous pressure in normal individuals are numerous, but the more important ones are posture, muscular activity, and respiratory activity. Because these introduce considerable variation, it is important for clinical purposes that the so-called normal venous pressure be established under conditions in which these variable factors are as far as possible controlled. This comprises what may be designated as basal conditions, and is strictly comparable to the clinical usage in the determination of oxidative metabolism. So far as venous pressure is concerned, basal conditions consist in rest in the recumbent or semi-recumbent position for a period of at least fifteen minutes be-before determination is made. Food ingestion or absorption, contrary to its well-known influence on metabolism, exerts no demonstrable influence on venous pressure, and may therefore be ignored. Under basal conditions in normal individuals, venous pressure, as measured in a superficial vein, is usually found to be between 40 and 60 mms. of water. In hundreds of observations made on hospital patients

in bed for rest treatment or minor surgical conditions, the highest venous pressure recorded in any case was 110 mms. of water (2). This may then be regarded as the upper range of normal.

The influence of posture is marked. Under conditions in which posture is changed without muscular exertion by the use of an adjustable table (48), venous pressure rises definitely in going from the horizontal to the erect position, and readings as high as 180 mms. have been obtained under the latter condition. The influence of age appears to be closely related to that of posture. In the recumbent position age has little or no influence, while in the erect (sitting) position the pressure rises progressively with age (32), in the later periods of life even exceeding that ordinarily found in decompensated patients at bed rest. The usual venous pressure found in cardiac decompensation, approximately 200 mms., is indeed the average that has been found in the sitting position in the normal subjects in the period between 45 and 55 years of age; an age group represented by many cardiac cases that become decompensated. In a series of 24 normal subjects,* varying in age between 9 and 61 years, the average venous pressure in the sitting position was 74 mms., falling to 50 mms. in the recumbent position. The highest readings observed were 100 and 80 mms.,

* The writer wishes to thank Dr. Middleton and Dr. Meyer of the staff of the Wisconsin General Hospital for the privilege of quoting these data, and also the readings in Cheyne Stokes respiration, in arterial hypertension, and in anesthesia.

respectively. The older subjects showed usually a higher venous pressure in the sitting position than the younger subjects, but no such difference was evident in the recumbent position. It is thus clear that the venous pressure may rise, under conditions present in the normal, equal to, or in excess of, that encountered in severe cardiac failure. This serves to emphasize the importance of establishing the normal when these conditions are controlled, and of always measuring venous pressure for clinical purposes under such conditions of control (basal).

In addition to the effect of posture, muscular activity may exert a considerable influence on venous pressure. Activity in a group of muscles is associated with vasodilation of the vessels in the active muscles, increased venous return to the heart, and increased output of blood from the heart into the arterial system. With each contraction of the muscles the venous return is also facilitated mechanically by direct pressure of the contracting muscle on the vessels. There is a play of antagonistic factors in this instance. The activity of the muscle increases the return of venous blood, and tends to cause a rise of venous pressure. The increased venous pressure leads to increased heart filling under a higher initial pressure and a consequent increased heart output; the latter of course acting to limit the rise of venous pressure. Another factor acting to

limit the rise of pressure in the peripheral veins is probably to be found in the increased aspiratory action of the thorax, associated with increased respiratory activity during muscular exercise (49). In most instances the influence of increased venous flow predominates, and the pressure rises (51). The extent of the rise varies with the severity of the exercise, and reaches its maximum when the exercise approaches the limit of effort of the individual (51).

The influence of respiratory activity is significant, not only because of its very definite effect under certain conditions, but also because it cannot always be controlled, or may depart considerably from the normal in disease. In cardiac decompensation, respiratory activity is nearly always increased, and it is necessary, therefore, to enquire carefully into the effect upon venous pressure that our present knowledge of this factor would lead us to expect. The negative pressure in the thorax facilitates the flow of blood in the venae cavae and determines a higher feeding pressure to the heart (pressure in right auricle) than exists in the veins outside of the thorax. The varying negative pressures in the thorax, associated with the different phases of respiration, must produce a correspondingly varying effect upon venous inflow. This must be greater, the deeper the respiration. The inflow is most facilitated during inspiration, due to the lower intrathoracic pressure, and the effect is proportional to the extent of inspiration.

During inspiration, the resistance to flow in the pulmonary circuit is reduced, facilitating the emptying of the right heart, increasing the inflow of blood into the right auricle, and causing a fall of pressure in the vena cava (52). Under conditions of quiet breathing in man, however, it has been found that so far as the pressure in the peripheral veins is concerned, there is little variation between the phases of inspiration and expiration. This is due to absorption of the differences encountered in the thorax by the inertia of the blood column between the central and peripheral veins (54). Definite respiratory variations in pressure in a peripheral vein in quiet breathing are thus absent or small (45) (46) (54). In hyperpnea, or overventilation, respiratory variations become evident, and there is also a fall of mean venous pressure, amounting in the normal subject to 7 to 30 mms. of water (45) (53), or even to as much as 110 mms. with excessive overventilation (55). In excessive overventilation it is probable that a part of the fall is due to reduction of the carbon dioxide content of the blood, in addition to the mechanical effect of lowered intrathoracic pressure (55). The former factor presumably results in an increase in the content or volume of the peripheral vascular bed by a general reduction of tonus of the vessels due to low carbon dioxide. The important point in reference to venous pressure in cardiac decompensation would appear to be that the accompanying overventilation would tend in itself to lower venous pres-

sure, and cannot in any way be responsible for the high venous pressure invariably found in this condition.

The influence of respiration on venous pressure is well illustrated by the variation in pressure in periodic breathing of the Cheyne-Stokes type. In recent observations in eleven cases, not as yet published, and made by Middleton and Meyer in the wards of the Wisconsin General Hospital, the average venous pressure during the periods of hyperpnea was 110, rising to 180 mms. of water during the apneic phases. Six of these cases were associated with signs of cardiac failure.

In the extreme variations in intrathoracic pressure produced during the Valsalva experiment (expiratory effort with the chest in the inspiratory position and glottis closed), and during the Mueller experiment (inspiratory effort with the chest in the expiratory position and glottis closed), extreme changes of peripheral venous pressure may occur. The extremely high venous pressures are encountered in the Valsalva experiment [420 mms. by Moritz and v. Tabora (37), 400 mms. by Kroetz (53), 500 mms. by Burger (56), and by Middleton and Meyer], while in the Mueller experiment the peripheral venous pressure appears to fall at first due to the extremely low intrathoracic pressure. In the Valsalva experiment, the extensive rise of peripheral venous pressure is due to the prevention of entrance of blood into the thorax, because of the positive pres-

sure in this cavity. With the lungs full of air, closed off from the outside and pressed upon by the contracting muscles, the normally negative intrathoracic pressure is replaced by a pressure of high positive value. The venous blood returning from the systemic peripheral vessels is thus blocked off from the thoracic venæ cavæ, and the peripheral veins become engorged with blood under high pressure. The amount of blood returning to the heart may be so reduced that the systolic arterial pressure falls to as low as 50 mms. of mercury. The venous pressure continues to rise as long as the respiratory effort is maintained. With the release of contraction of the thoracic muscles, the engorged veins discharge into the thorax, and the heart is subjected to a large amount of venous blood under a high pressure. The normal heart responds quickly with increased output, and systolic arterial pressure may rise to 250 mms. of mercury, or more, in normal subjects (57). The abnormal states of intrathoracic pressure, and their influence on venous pressure, are of further significance in illustrating the ability of the normal heart to react to a large but transitory venous overload, and to increase its work as a result. On the other hand, a smaller overload may result in heart failure, in a heart in which the ability to respond is reduced by disease. The Valsalva experiment is a duplicate of many efforts of strain, and it is well known that these may mark the onset of partial or complete heart failure in subjects with organic heart

disease. The lifting of heavy weights, sudden violent muscular efforts, and straining at stool are practical duplicates of the Valsalva experiment with its attending alterations in venous pressure, and venous load upon the heart.

CHAPTER V

Venous Pressure in Cardiac Decompensation

The physical alterations present in the circulation when the heart fails to respond adequately to the venous load have been considered in some detail. It is obvious that in this condition the venous pressure must be high as compared with the basal normal. To this conclusion nearly all observers who have recorded venous pressure in cardiac decompensation agree. The first observation of this sort was made by Frey (30) in 1902, and by Gaertner (34) a year later. In 1908, Hooker and Eyster (32) made numerous readings in several cases of decompensation, and showed that the venous pressure rise was, in general, related to the severity of the clinical condition, and that restoration of compensation was associated with a fall of pressure to normal. Clark (23), in 1914, made the first detailed studies of a considerable number of cases, and emphasized the value of the determination of venous pressure in the therapy and prognosis of this condition. Clark established the "critical level" of venous pressure in decompensation at 200 mms. of water. Above this level a rising venous pressure means a rapidly failing heart, while a falling venous pressure is of favor-

able diagnostic import. Eyster and Middleton (2), in 1924, found an invariable rise of venous pressure when signs and symptoms of cardiac decompensation were present, and further showed that a general rise of venous pressure was encountered clinically only in this condition. During this period a number of other reports appeared in the literature dealing with a few cases, or with single observations of venous pressure on individual cases. Nearly all of them confirmed the conclusion that venous pressure is high in decompensation, and falls to normal as compensation is restored. The extensive and detailed observations were all made by the indirect method, since the direct cannulation of the vein permits of only occasional measurements. However, the general conclusion was sustained by several workers using the direct method. The only dissension from the complete acceptance of this conclusion has come from a few observers employing direct cannulation of the vein. Ratky and Klein (58) found an invariable rise of venous pressure above the normal level in cardiac decompensation, but also in certain other conditions, and concluded that it was not pathognomonic of heart failure. They also expressed doubt that the extent of the rise is paralleled by the clinical severity of the case. Kroetz (53) concluded that while venous pressure was elevated in severe decompensation, mild cases were observed with a normal pressure, and that the elevation is not always proportional to the degree of failure.

Relation to Clinical Course. Routine observations of venous pressure in cardiac decompensation, if made daily or at more frequent intervals, show usually a direct relationship to the clinical condition of the patient. The actual height of the venous pressure, unless unusually high, does not appear to be as important as the trend. The venous pressure, in our experience, is always elevated above the basal normal in definite decompensation when the patient first comes under observation. The initial height, while perhaps suggestive regarding the severity of the failure, unless it is in the extreme upper ranges encountered clinically, appears to be of no great significance in prognosis. This depends much more upon the course of the venous pressure within the early days of treatment. In favorable cases, the venous pressure falls rapidly from its initial high point to reach only a slight elevation, or even normal, within a few days. In cases with water retention this is associated with a marked diuresis. Signs of congestive heart failure gradually disappear, and there is a marked and often astonishing improvement in subjective symptoms. The diuresis, relief from congestion, improvement of symptoms, and fall of venous pressure appear in most cases to proceed together, but frequent and careful venous pressure readings in many instances will indicate that the fall of venous pressure precedes, as would be expected, the improvement in kidney function, the unloading of water, and the reduction in vascu-

lar congestion. In the less favorable case, perhaps with no higher or even lower initial pressure, the fall is slower, and a pressure is maintained with frequent variations well above normal for weeks, or even occasionally for months. The fluctuating venous pressure during this period appears to be closely associated with the varying signs of congestion, often with the symptoms of the patient. If carefully and frequently determined, it may be shown to definitely precede the changes in signs and symptoms. During this more or less prolonged period, often for no apparent reason, there may occur a rather rapid rise of venous pressure with a negative water balance and increase in signs of congestion and symptoms, perhaps disappearing in a few days, or proceeding to a fatal termination. Other cases are encountered, usually with a high initial venous pressure, in which treatment produces no change in the venous pressure, or only a slight and often temporary fall. Death ensues in these cases invariably within a short period. Finally, in a few cases there is a prompt response to treatment; the clinical condition improves greatly, and restoration of compensation appears imminent. The venous pressure falls frequently to within the normal range. Complete restoration, however, does not occur. With efforts to get the patient upon any reduction in the strict régime of treatment, or even without obvious cause, the venous pressure rises, signs of congestion increase, and the clinical condition be-

comes worse. Cases of this type represent a myocardium so badly injured as to offer little chance of improvement to the point where it can handle more than the load placed upon it, when this load is reduced to a minimum by protective treatment.

There is every evidence to lead to the belief that in the decompensated heart two primary factors must be considered: the functional capacity of the heart muscle, and the load upon this muscle as represented by the venous pressure. It is most probable that the normal heart muscle can react effectively to any load that can be put upon it, except under extreme experimental conditions. If of a transient character, this load may be in excess of that encountered in clinical cardiac failure in organic heart disease. As already stated, pressures as high as 500 mms. of water in the peripheral veins may be encountered in the Valsalva experiment, and in severe muscular exercise the venous pressure may rise higher than found in all but the exceptional case of cardiac decompensation. It would appear that in man, decompensation is invariably accompanied by the other factor: a decrease in the functional capacity of the heart. This is most frequently the result of long-existing organic disease, or less frequently, probably, from toxemia, as appears to be true in the failure occurring in lobar pneumonia, diphtheria, and occasionally in other severe acute infections. The heart, in which the ability to respond to increased load is thus reduced, is liable to failure at any time. This

failure is associated with the upper normal variations of venous pressure, resulting from some unusual physical strain or other factor. It is possible, in many clinical cases of organic heart disease in their first decompensation, to learn of some activity or strain which can be held immediately accountable for the onset of the failure. Not infrequently the initial failure in chronic heart disease results from the sudden onset of auricular fibrillation. The reduced mechanical efficiency resulting from this (59) is sufficient to narrow the range of response of the muscle to below the existing venous load, and decompensation is established. There is reason to believe that with the overload and stretching of the muscle that occurs in decompensation, further permanent injury and further reduction of the functional capacity of the heart muscle results, and subsequent failures may occur with a less excessive load. Finally, the condition may be reached in which the muscle can handle the load only when at its minimum, or in other words, only when the venous pressure is maintained at its basal level by complete physical inactivity. Usually, however, before this stage is reached, the load overcomes the inefficient heart muscles, and a fatal decompensation supervenes.

The cardiac muscle factor is emphasized by the relation of age to cardiac decompensation. The great majority of instances of decompensation are encountered in two age periods; between 35 and 50

and in extreme old age. The patients of the former group have in most instances hearts which have been injured by acute infectious disease, particularly rheumatic endocarditis or syphilis. The latter group consists of patients with primary myocardiac injury due to coronary sclerosis, or to the changes associated with hypertrophy from arterial hypertension. The actual initial damage to the heart in the first group in most cases occurred years previously. Acute rheumatic fever is most commonly encountered in young adults between the ages of fifteen and thirty years. While rheumatic endocarditis in certain cases undoubtedly reaches a slowly progressing chronic stage with occasional exacerbations and a slowly progressing injury, in most cases the process is self-limited, and the initial damage is the total damage caused by the infection. As a result of the initial valve injury, a mechanical handicap results. This leads at first to dilatation or stretching of the muscle (60). Recent experimental work has shown that this stretching results in injury to the muscle fibers (61), and that the hypertrophy that follows this injury is a response to the injury rather than to the increased work of the muscle alone. Compensatory hypertrophy develops, and for a period, usually for years, circulation is maintained at its normal level, and the ability of the heart to respond to the usual increased load encountered during normal physical life, or even to excessive loads, is restored. Extensive valvular lesions with marked cardiac

muscle hypertrophy may be found in the second, or even the third decades, of life to be compatible with normal, or even excessive, physical activity, and there is apparent little if any narrowing of the ability of response on the part of the heart to increased load. This has been proven to be true also in more or less accurately measured overloads applied to experimental hypertrophies produced in laboratory animals (62) (63). The hypertrophied muscle, provided the lesion is not too extensive, thus restores, or nearly restores, the normal range of response in spite of the mechanical defect in the heart as a pump. The hypertrophic heart muscle appears, however, to be more prone to early degenerative changes than the normal muscle, and in the usual case of chronic valvular heart disease, evidences of this occur during the third or fourth decade of life. In the absence of an addition to the initial damage by a repetition or chronicity of the infectious process, compensation is well maintained until this factor becomes manifest. With it, the ability of the heart muscle to respond to the higher venous loads encountered normally is reduced, and decompensation ultimately supervenes.

Relation of venous pressure to the water balance. Water is normally lost from the body through the urine, skin, expired air, and feces. The exact water balance of the body should include a comparison of the total water loss with the total intake, but it is customary to consider clinically only that lost

through the urine. The proportion of this to the total will obviously vary in a normal individual with a number of factors, such as the extent of loss through the skin as influenced by temperature. In disease, a number of new factors may enter: alteration in the fluidity of the stools, vomiting, hyperpnea, and alterations in kidney function. If the water balance is taken as the relation between fluid intake and urine output, it is obvious that this will be positive under normal conditions, i.e., more fluid will be taken into the body than appears in the urine, since some is lost through other channels. The normal relation between fluid intake and urine secretion shows considerable variation when a group of subjects is kept under approximately standard conditions on a fixed fluid intake, and indeed the same individual may show considerable variation from time to time (64). A consideration of the results from numerous determinations (65) indicates that under normal conditions the urine represents from 54 to 65 per cent of the total fluid intake, with an approximate average of 60 per cent. The intake ordinarily varies between 2300 and 3700 cc.; the urine between 1500 and 2000 cc. in 24 hours. Any definite increase of the percentage of the urine output above 60 per cent may be termed diuresis. Under these conditions more water is being lost from the body than is being taken in, and a negative water balance results. Just when the negative balance is established is impossible to say when one is

measuring only the urine output, but the increase in urine may be sufficient to leave no doubt that it exists, and in marked diuresis, the water lost through the urine may alone exceed the total intake. Under favorable conditions in an edematous individual, the urine may be several times the water intake.

It is believed that water is normally held in the blood mainly through the osmotic pressure of the blood proteins (colloids). Although many other materials are present which might be supposed to alter the osmotic pressure, all of these, with the exception of the colloids, are readily diffusible. In order to maintain an equilibrium of osmotic pressure, substances which are non-diffusible, or which diffuse with extreme slowness, are alone to be considered. The osmotic pressure of the plasma colloids under average normal conditions is approximately 14 mms. of mercury (66). Water tends to leave the blood mainly under normal conditions through the process of filtration. The extent of this force is determined by the physical pressure of the blood in the capillaries. The average capillary pressure in the skin capillaries in man has been estimated by indirect determination at approximately 15 mms. of mercury under basal conditions (2), rising somewhat in the erect position, and as a result of physical activity. Micro-cannulation of the capillaries in the experimental animal, with direct measurement of pressure, shows that there is a definite fall in pres-

sure from the arterial to the venous end of the capillary. At the arterial end, the pressure exceeds, while at the venous end, it is lower than the osmotic pressure of the plasma colloids (67). It appears, therefore, that fluid and dissolved materials may under usual conditions leave the arterial end of the capillaries by filtration, while water is being absorbed into the venous end by osmosis. This appears to help to maintain the blood volume, and at the same time to facilitate the passage out of dissolved materials. Any increase in the water content of the blood, without a simultaneous increase of the colloid, results in a prompt loss of the excess water. Even with the simultaneous injection of diffusible materials, such as sodium chloride or glucose in sufficient concentration to momentarily raise the osmotic pressure of the blood, osmotic equilibrium is restored within one or two minutes, and the excess water leaves the blood within a half hour. If a colloidal substance such as gum acacia is injected with the water, the rise of osmotic pressure is maintained longer, due to the low diffusibility of the material. The excess water may remain in the blood for six hours or more (68). The blood volume, and the water equilibrium between the blood and tissues, is maintained under normal conditions within narrow limits, and any excess of water entering the blood stream is promptly excreted by the kidneys. In the modern conception of kidney function, it appears that water excretion occurs by filtration in the glo-

merulus, and is a mechanical process with no evidence of increased functional activity on the part of the kidney. It is presumably determined, therefore, by the relation between the physical pressure of the blood in the glomerular capillaries, and the osmotic pressure of the plasma colloid. Actual physiological work on the part of the kidney, as indicated by an increase in oxygen absorption and carbon dioxide elimination by the organ, is confined to the excretion of certain dissolved material from the plasma, and the resorption of water, probably by the tubules, resulting in the concentration of the urine as finally excreted into the pelvis. In the diuresis established from excessive water intake or from the loss of excess water from edematous tissues, there is no evidence that actual increased work on the part of the kidney occurs, and the process is explicable on purely mechanical grounds.

Excessive loss of water from the blood, and its accumulation in tissues and serous cavities, is a common accompaniment of cardiac decompensation, and is responsible for many of the signs and symptoms that accompany this condition. It is ordinarily supposed that this is due to the excessive filtration pressure in the capillaries, as a result of the engorgement and rise of pressures in these vessels. That the capillary pressure is high in heart failure has been demonstrated (69), and is unquestionably due to the high venous pressure with unchanged or increased arterial pressure. The capil-

lary pressure under these conditions must vary directly with the pressure of the blood in the arteries and veins. Capillary pressures as high as 30 millimeters of mercury are commonly encountered in cardiac decompensation. As edema becomes established in the surrounding tissues, the turgor that results tends to oppose further filtration, and thus a factor comes into play tending to limit further accumulation of excess water in the tissues. This factor is least in the tissues normally under small elastic tension, and is of course absent on serous membrane surfaces. Another factor influencing the edema through its effect on capillary pressure is gravity, and this is ordinarily manifested especially in the feet and legs, where the difference in level between this region and the heart retards the return flow of venous blood, and results in a high venous and capillary pressure. Probably other factors play a rôle in the excess passage of water from the blood into the tissues and serous cavities. In addition to the high capillary pressure in cardiac decompensation, direct observations show that the flow is retarded, and the circulation time through the capillaries is increased (70) (2). This results in an excessive reduction in the oxygen content of the blood in this region of the vascular system. Normally, the venous blood contains approximately 15 volumes of oxygen to each 100 cc. of blood, having lost about 5 volumes per cent in the capillaries (71), while

in decompensation the oxygen of venous blood may be reduced to 5 volumes per cent or even lower (72). The oxygen content of the blood entering the capillaries from the arterioles may also be reduced due to deficient aeration in the lungs (73). The capillaries as well as the tissues are limited in their oxygen supply due to the reduced volume of blood passing through them in a unit of time; the result of a slowing down of the whole circulation (20) through diminished cardiac output and peripheral engorgement. That anoxemia leads to increased permeability of the capillary wall, and causes an increased passage of fluid, even with unchanged capillary pressures, is probable from direct experimental study (74). The degree of this influence on different capillary regions may perhaps explain in part the greater passage of water in certain regions than in others, independent of the other factors considered. In cardiac decompensation we thus have an engorged peripheral circulation, associated with high capillary pressure and reduced capillary flow, and increased permeability of the capillary wall due to deficient oxygen supply. This results in a disturbance of the normal balance between the water of the blood plasma and the water content of the surrounding tissue spaces and cells, so that an excessive accumulation occurs in the latter. The extent of this in different regions is probably determined by local variations in the factors concerned in its production.

The reduced kidney activity commonly found in cardiac decompensation has been generally referred to the reduced circulation in the kidney, and the high venous pressure that results. Actual mechanical interference with the flow of urine in the tubules has been ascribed to the engorged interlobular veins, although the modern view is that the obstruction probably occurs in the glomerulus. Experimental work in reference to the mechanism of urine secretion has emphasized two main factors in reference at least to the amount of water secreted: capillary pressure (filtration pressure), and capillary flow. Changes in pressure alone, with constant blood flow, produce parallel changes in urine formation (75). Marked reduction of blood flow, even in the presence of a high capillary pressure, results in a lessened excretion, whether due secondarily to mechanical obstruction, or primarily to reduced volume of blood in a unit of time.

It is evident that one of the essential and perhaps the most essential factor in the maintenance of the usual distribution of water between blood and tissues and in the formation of urine is the capillary pressure. It is further evident that the increased capillary pressure and stasis which exists in cardiac incompetence results from the inability of the heart to empty adequately the venous reservoirs, and is associated with, and goes parallel with, a high venous pressure. The rise of capillary and venous pressure must precede the excessive passage of fluid

out into the tissues, and the reduced urine secretion with water retention. A fall of venous and capillary pressure must likewise precede the restoration of kidney function and the unloading of the excess water. With the improvement in cardiac activity associated with treatment, the heart more adequately empties the large veins, and the venous pressure, and consequently the high filtration pressure in the capillaries, is reduced. Coincidentally, the circulation is accelerated, and more blood passes in a unit of time through the peripheral vascular circuit. Anoxemia is reduced, kidney function restored, and water begins to pass back from the engorged tissues into the blood as excretion of water continues. The rapidity of unloading of water and the restoration of a normal water content and distribution is frequently one of the most astonishing evidences of therapy that occurs in the whole field of medicine.

That there is a definite relation between venous pressure and urine secretion in cardiac decompensation was first established by Clark (23). The urine output in relation to the fluid intake varies approximately inversely as the venous pressure, and if the pressure determinations are made frequently enough, it is possible to show in many cases that the changes in venous pressure precede the changes in urine output. This inverse ratio is not always evident, and there are probably other factors concerned, but it is usually present and frequently striking. A record

of the water balance is now recognized as an important routine in all cases of cardiac decompensation, and is of undoubted value in controlling therapy. The careful and frequent determination of venous pressure is also of importance as it is indicative of impending changes in the extent of edema and kidney function in many cases, even before the change is actually established. A number of charts showing data concerning the water balance and venous pressure are to be found in Chapter VII.

The water balance in 108 cases of cardiac incompetence from the wards of the Wisconsin General Hospital has been recently studied by Van Valzah, McKinley, and Middleton (76). They are classified as follows:

	Surviving	*Dying*
Edematous	46	45
Non-edematous	13	4
	59	49

The data show that the great majority of all cases in the edematous group develop a primary diuresis under hospital treatment. This tendency to diuresis is less marked and less sustained in cases proceeding to a fatal termination, and occasionally is absent. Usually the surviving cases show a reduced urine output after the primary diuresis, but an early recovery occurs with a satisfactory output intake ratio. These patients were at bed rest, received from 1000 to 1500 cc. of fluid daily on admission, and as other articles of diet were added, the salt

was kept at a minimum until the edema disappeared. Digitalis was used routinely, and in certain cases xanthin diuretics, calomel, and novarsurol were administered when diuresis did not occur promptly. Occasionally, striking improvements were found to occur after the removal of fluid accumulations from one or the other of the serous cavities. The inverse relation between venous pressure and water balance in certain cases was apparent.

The relation of venous pressure to cardiac dilatation in decompensation. The heart increases in size in the normal intact animal as the venous pressure is increased up to a certain maximum, which is represented by the capacity of the pericardial sac (77). The size of the heart in diastole appears to depend upon this factor alone, and is independent of the rate of contraction (78). The venous pressure determines the filling pressure in diastole, and the relaxed heart dilates in proportion to it.

It has long been believed by clinicians, a belief supported by evidence derived from topographic percussion, that cardiac incompetence is accompanied by diastolic distention, and that this condition is commonly associated with a dilated, weakly contracting heart which usually beats at a fast rate. The dilatation appears to involve especially the right auricle and right ventricle. Accurate topographic percussion of the heart, especially the right border, is generally recognized, however, as a difficult procedure, and this difficulty is emphasized

when, as so frequently occurs, there is accompanying pulmonary congestion and not infrequently hydrothorax. More accurate information regarding this problem may be obtained by roentgenological studies of the heart in decompensation. In Table I, heart-size measurements from six cases of cardiac decompensation, made at intervals throughout the period in which compensation is being restored, are compared with the venous pressures and the water balance.

The x-ray measurements include the area and greatest transverse diameter of the frontal plane teleroentgenograms. These were made with a bedside roentgen ray unit, the patient reclining at an angle of thirty degrees on a special back rest, and with precautions being used to secure accurate alignment of the tube and film. The distance from target to tube was two meters, and the measurements were corrected for distortion. The first two cases, for which data are given in the table, are of mild decompensation with only moderate elevation of venous pressure. During restoration of compensation the venous pressure fell from 120 to 80 mms. in the first, and from 100 to 50 mms. of water in the second. No significant changes were observed in the area or transverse diameter of the cardiac silhouette. The remaining cases were of more severe decompensation with much higher initial pressure. Case 3 showed a reduction of 16 per cent in area, and 3 per cent in transverse diameter.

TABLE I

Venous Pressure and Heart Size in Cardiac Decompensation

Case	Disease	Days in Hospital	Water Intake in Oz.	Urine Output in Oz.	Venous Pressure in cm. of Water	Teleradiogram		Change in Per Cent of Preceding Determination	
						Area in Sq. Cm.	Transverse Diameter in Mm.	In Area	In Tr. Diameter
1	Mitral stenosis, mitral regurgitation, auricular fibrillation	1	14	33	12	202	163	...	...
		2	20	30	11	206	160	+2	−2
		5	30	35	10	206	160	0	0
		8	30	31	6	209	160	+2	0
		9	..	..	8	198	161	−5	+1
2	Mitral stenosis, mitral regurgitation	2	40	25	10	185	159	...	...
		3	55	30	7	187	153	+1	−4
		8	35	35	5	183	160	−2	+4
3	Mitral regurgitation, mitral stenosis, auricular fibrillation	1	42	6	23	304	200	...	...
		6	52	19	14	268	190	−11	−5
		15	38	19	18	265	190	−2	0
		25	28	28	8	256	193	−4	+2
4	Mitral stenosis	2	35	10	23	190	136	...	...
		6	37	22	13	193	141	+2	+4
		13	30	32	9	170	126	−12	−11
5	Mitral regurgitation	1	..	..	24	227	188	...	...
		2	46	4	17	217	186	−4.5	−1
		3	31	18	15	221	185	+2	0
		4	38	68	12	211	178	−5	−4
		5	36	44	9	217	180	+3	+1
		10	37	36	10	211	167	−3	−7
6	Mitral stenosis, mitral regurgitation, auricular fibrillation	1	33	7	23	221	164	...	...
		3	40	20	20	222	171	0	+4
		6	45	12	13	227	172	+3	+1
		15	30	35	7	218	163	−4	−5
		40	37	37	7	229	169	+5	+2
		55	30	17	17	238	173	+4	+3
		57	25	25	8	210	157	−12	−9

Case 4 showed a 10 per cent reduction in area and a 7 per cent reduction in transverse diameter. In case 6 there were two well-marked periods of decompensation as determined clinically. In the first period the venous pressure fell from 230 to 70 mms. The area and transverse diameter measurements failed to reveal significant changes. In the second period of decompensation the venous pressure fell from 170 to 80 mms. There was a reduction of 12 per cent in area and 9 per cent in transverse diameter. The changes in area are relatively small, but reach considerable magnitude if calculated as heart volume. If, for example, the difference between the largest measurement recorded in case 6 is compared with the smallest measurement in this case, and the corresponding volumes of the heart calculated in each case from the area of the teleroentgenogram by the method of Bardeen * (79), the large volume with a venous pressure of 170 mms. is 1950 cc., the smaller with a venous pressure of 80 is 1675 cc.; a difference of 275 cc., or approximately 14 per cent reduction. It would appear from these results that as the decompensated heart regains compensation, there usually occurs, along with the falling venous pressure, a reduction in diastolic size or volume of the heart. These changes

* Bardeen's formula for estimating heart volume from the area of the teleroentgenogram is Volume $= 0.53\ \text{Area}^{3/2}$. The approximation so obtained has been proven by control measurements on cadavers, and lies within $\pm$ 10 per cent variation from the actual.

are not marked, however, and it is questionable whether they would be accurately revealed by percussion. Percussion of the right cardiac border during decompensation in an effort to estimate the progress of restoration would certainly appear to be of doubtful value. A much more accurate estimate of the distending diastolic load is revealed by measurement of the venous pressure.

The capillary pressure and flow in cardiac decompensation and its relation to venous pressure in this and in other conditions. That the venous pressure is largely independent of capillary pressure in the normal subject, or indeed in various conditions other than heart failure, is indicated by a number of observations. Local changes in vascular tonus, produced by warming or cooling the hands, cause no significant changes in venous pressure, although the volume of the veins is markedly affected (16) (35). In the extreme local stagnation of the peripheral circulation encountered in the "Raynaud Phenomenon," very slight or no changes in pressure occur in the medium-sized veins. In extreme cases, the veins may be much reduced in volume, but with unchanged pressure (17).

The relative independence of venous and capillary pressures, so long as cardiac function is normal, is evident from a study of the pressures in conditions of lowered tonus of the vessels in the peripheral vascular circuit. Many acute febrile conditions

show evidence of low peripheral vasomotor tone, with normal pressures, however, in the peripheral veins (2). This condition is especially well developed in acute rheumatic fever, and observations made in a series of ten cases of this disease (2) are summarized in Table II. In these cases, in addition to the measurement of arterial, capillary, and venous pressures, observations of capillary flow and of gross alterations in the peripheral circulation were made. The capillary pressures were determined by the apparatus and method of Danzer and Hooker (80), which allows direct observation of the capillary loops in the finger-nail bed through a microscope at a time when they are being submitted to outside pressure. Observations on a series of normal subjects at rest in the recumbent position showed an average capillary pressure of 15.6 mms. of mercury. The average pressure in the cases of acute rheumatic fever was somewhat lower. In the microscopic field, employing this method of observation, one ordinarily sees from twelve to fourteen capillary loops. In normal subjects, corpuscular flow is present in only one-half to three-quarters of the capillaries visible. A higher percentage of patency and flow exists in the dilated capillaries of acute rheumatic fever. Other evidences of low vasomotor tonus in this series of cases are the low diastolic pressure, high pulse pressures, macroscopic capillary pulse present in all but two, and auscultatory sounds over the peripheral arteries, present in all but one case. From

TABLE II

ACUTE RHEUMATIC FEVER

Case	*Duration of Observation in Days*	*Arterial Pressure in mms. of Mercury*			*Venous Pressure in mms. of Water*			*Average Capillary Pressure in mms. of Mercury*
		Systolic	*Diastolic*	*Pulse Pressure*	*High*	*Low*	*Average*	
1	29	113	57	56	30	60	40	9
2	102	127	60	56	70	50	60	25
3	43	125	64	61	80	50	60	19
4	31	113	64	49	50	30	40	12
5	22	110	65	45	50	30	40	9
6	15	105	65	40	80	40	60	11
7	19	135	70	65	80	30	60	13
8	23	118	74	44	80	30	50	12
9	80	116	82	34	80	30	60	13
10	20	116	58	58	70	30	40	13
Average	38.4	118	66	58	67	38	51	13.6

the experimental side, the extensive work of Plumier (81) demonstrated the relative independence of arterial and venous pressures and the absence of any effect on the latter of peripheral vasomotor changes, unless cardiac activity was altered. These and other observations point to the conclusion that venous pressure is to a large degree independent of the amount of blood coming into the veins through the capillaries, or the pressure of the blood in these vessels or in the arteries, so long as the heart is competent. Venous pressure, since it is the main factor that determines the extent of cardiac work, is in itself largely regulated and maintained within limits by the response that it calls forth from the heart. Any increased flow with a tendency to a rise of pressure results quickly in increased heart output, and in this way any considerable accumulation of blood, or rise of pressure in the veins, is prevented.

Quite different, however, may be the influence of a high venous pressure on capillary flow and pressure in the presence of a heart unable to respond adequately to the abnormal venous pressure. The venous stasis leads to capillary stasis and engorgement, and a rise of capillary pressure (82) (83) (69) (84). In decompensation associated with relative tricuspid insufficiency, an actual retrograde flow of corpuscles may be observed, and collisions may occur between corpuscles coming from the arterial limits of the capillaries and others regurgitating

from the venules (85). The latter are obviously the result of venous stasis and regurgitation attending the tricuspid insufficiency. If the determination of capillary pressure and observation of capillary flow were not attended with considerable technical difficulty, the method would be of value clinically in estimating the degree of vascular stasis in decompensation. Observations such as those quoted in case VI, Chapter VII, indicate the close relationship that exists between capillary flow and the clinical state. They also indicate, however, the close relationship existing between the capillary and venous circulations in decompensation. Measurement of venous pressure, which can be done so much more readily, gives the same information. It should be mentioned that the older and somewhat simpler method, applied in an effort to measure capillary pressure, and consisting in determining the amount of pressure required to blanch the skin (86) (1), probably does not measure the capillary pressure, but gives an approximation of the pressure in the smaller venous plexuses. It is these vessels which cause the normal color of the skin (80) (35). Finally, it is the stasis and engorgement of these vessels which account, in part at least, for the cyanosis, independent of changes in oxygen content, which occurs in conditions of high venous pressure (87).

The effect of therapy on venous pressure in cardiac decompensation. The venous pressure in a peripheral vein reaches its lowest value with the

subject in the recumbent or semi-recumbent position, and in a state of muscular rest. In a heart laboring under a venous load which is beyond the physiological limit of response of the cardiac muscle, the first and paramount therapeutic effort should be to reduce the load to a minimum. In addition to the mechanical effects of rest and posture, oxidations in the peripheral tissues, and especially the muscles, are reduced to their maintenance value, and the tendency to anoxemia reduced. The importance of this latter factor upon the heart muscle, and the maintenance of capillary integrity have already been considered.

There is some evidence to indicate that in certain normal subjects, particularly in women, the volume output of the heart may be reduced in the semi-recumbent and erect posture as compared with the recumbent posture (88) (89) (19a) (90). These observations are based on the gasometric determination of cardiac minute volume; a method which is open to rather serious question as to its reliability when applied to man (91) (92). They also show considerable discrepancy (90) (94). The greatest and most constant difference that has been found is the diminution in output on standing. A reduced return of venous blood to the heart, due to stasis in the vascular beds of the lower extremities and splanchnic areas resulting from gravity, is the explanation that has been given for this (93) (89). It is difficult to reconcile this explanation in the usual

normal subject with the higher venous pressure that results on assuming the erect posture, although the effect of long standing without muscular activity has long been known to result, in certain subjects, in vascular engorgement of the lower extremities, and in reduction of blood flow to the brain and upper extremities. Diminished vascular tonus under these conditions is more commonly encountered in women, and reaches its most marked development in certain cases of neuro-circulatory asthenia, where fainting attacks are not uncommon, and are presumably due to reduction in blood flow to the cerebrum. Differences in individual vasomotor reaction to the effects of gravity may in part explain the observed discrepancies regarding heart output. It has long been known that many patients suffering from congestive heart failure are more comfortable and in better clinical condition when elevated to a semi-recumbent or sitting position. Field and Bock (89) have ascribed this to the effect of gravity in lessening the return flow of venous blood to the heart, and a consequent reduction in pulmonary congestion.* They state that it may be equivalent to venesection insofar as the effect on the circulation in the lungs is concerned. In considering the possible effect of

* This suggests that perhaps the venous pressure change on changing posture may be different in cardiac decompensation from that which is usually found in the normal, and in two cases of cardiac decompensation with orthopnea, Dr. W. S. Middleton reports a fall of venous pressure from 340 to 230 mms. and from 140 to 70 mms. of water respectively, on changing from the recumbent to the sitting position.

gravity in reducing the return flow of venous blood, it should be noted that so far as the effect on cardiac output is concerned, it should be opposite in the normal and in the decompensated heart. In the former a reduction of return flow would reduce the minute volume output, while in decompensation a reduced return flow might well lead to an increase in cardiac efficiency, and in the amount of blood pumped. If the reduction of venous load is sufficient, in favorable cases it is conceivable that it may be lowered to a point where it again comes within the range of physiological response of the cardiac muscle, or at least more nearly approaches it. This must result in an increased output and a still further reduction of venous load. Unfortunately, the methods so far devised for studying heart output in man are ill adapted to conditions existing in dyspnea, and direct observational details are lacking in this as in many other conditions where such data are desirable.

One of the most important aspects of the effort to influence the circulation in a mechanical way to combat heart failure is blood letting or venesection. By removing a portion of the blood directly from the engorged venous bed, a direct effort can be made to lower the venous load upon the heart and perhaps reduce it, temporarily at least, to within the physiological limits of response of the cardiac muscle. Although it is probable that the general

blood volume is not increased in cardiac decompensation (95), the proportion of the total volume of blood in the veins is greater than normal. The attending vasoconstriction, by means of which a normal or increased arterial pressure is maintained in the presence of a diminished cardiac output, and the failure of the heart to move the usual amount of blood from the veins into the arteries, results in a stagnation of the blood in the veins, and a proportionate increase in volume in this part of the circulation. The effect of removing blood from the venous system is quite different in the normal subject and in congestive heart failure. In the former, both in the experimental animal and in man, moderate bleeding up to two per cent of the body weight results in only slight and transitory effects on venous pressure, arterial pressure, and heart volume (7) (9). In conditions of high venous pressure with cardiac decompensation, however, venesection usually produces a definite and often prolonged fall of venous pressure (32) (40) (96) (96a). This is associated with a decrease in heart size as determined roentgenographically (98). The venous load may thus be definitely reduced, and the diastolic distension or stretching of the heart under its influence lessened. It is conceivable that by this procedure a considerable and lasting improvement in the circulation may be effected, although from the mechanical standpoint alone, only a transitory

effect is probable. Even a brief lowering of the load to within the range of physiological response of the cardiac muscle must result in an immediate response of the heart with more efficient contractions, and this in turn to a rapid amelioration of the unfavorable conditions of high coronary venous pressure and anoxemia; conditions leading inevitably to lowering of the functional capacity of the muscle. If the ability of the heart to respond effectively is not too much reduced, and it is possible to get the venous pressure down to that level for a period which would otherwise be transitory, the heart may recover sufficiently to handle the load and prevent a further rise of venous pressure. The "vicious cycle" of cardiac incompetence, mounting venous pressure with failing heart muscle, and failing heart muscle leading to still further venous congestion and a still higher pressure, may be broken. If this cycle is once broken, improvement in the nutrition of the heart muscle, and its better ability to respond to the load to which it is exposed, result in restoration of a competent heart.

The writer, in collaboration with Dr. W. S. Middleton (97), has recently reviewed the results of venesection in twenty-one cases of congestive heart failure from the wards of the Wisconsin General Hospital. The clinical data on these cases is summarized in Table III, as shown on pages 72-73. The following comment and summary of this study is in part quoted from this report.

Two of the cases were in young adults, the rest of the patients ranged between thirty and seventy years of age. The basic lesion was approximately equally divided between primary endocarditis and hypertensive myocardial degeneration. Sixteen of the twenty-one patients gave a history of previous periods of decompensation. Immediate clinical improvement following the venesection occurred in fifteen of the twenty-one cases. The initial height of the venous pressure was apparently without significance as to whether improvement occurred or not. Of the six cases showing no favorable response to venesection, death occurred within forty-eight hours in four, and in ten and twenty days, respectively, in the remaining two. Thirteen of the cases died while still in the hospital. However, in those patients showing an immediately favorable response to venesection, the duration of life was longer, and it appears reasonable to regard them as having rallied from the emergency which led to the phlebotomy. Of the nine, Cases **2**, **3**, **6**, **7**, **10**, **11**, **14**, **18**, **21**, which survived as long as one week following venesection and later succumbed, seven showed early amelioration of symptoms, and four of the seven finally died of circulatory accidents, such as pulmonary infarction and cerebral hemorrhage. It should be noted that this group represents a series of cases of particularly severe decompensation, and in most cases venesection was not resorted to until other measures had failed. This would in large

TABLE III

Clinical Data on Venesected Cases

Case	Age	Sex	Basic Lesion	Etiology	Decompensated		V. P.	Amount Blood Let
					Remote	Present		
1	64	M.	Myocardial degeneration	Hypertension; arteriosclerosis	4 yrs.	Continued	21	400 cc.
2	62	M.	Myocardial degeneration	Hypertension; arteriosclerosis	7 mos.	1 mo.	20	350 cc.
3	62	F.	Endocarditis	Rheumatic fever; arteriosclerosis	7 yrs.	6 mos.	20	500 cc.
4	40	M.	Endocarditis	Rheumatic fever	3 yrs.	6 wks.	19–20	500 cc.
				Three Days Later			21	500 cc.
5	55	M.	Myocardial degeneration	Arteriosclerosis with coronary involvement	1 yr.	2 wks.	Edema	500 cc.
6	73	M.	Endocarditis	Rheumatic fever (?)	(?)	2 wks.	36	450 cc.
7	43	F.	Endocarditis	Scarlet fever		9 mos.	28	500 cc.
8	69	M.	Myocardial degeneration	Hypertension; arteriosclerosis	4 yrs.	2 mos.	26	500 cc.
9	17	M.	Adhesive pericarditis	Multiple serositis; rheumatic fever	1½ yrs.	6 mos.	20	300 cc.
10	51	M.	Endocarditis	Rheumatic fever (?)	2 yrs.	3 mos.	22	500 cc.
11	54	M.	Myocardial degeneration	Hypertension; arteriosclerosis	2 yrs.	Continued	Edema	600 cc.
12	34	F.	Aortitis; myocardial degeneration	Syphilis	7 mos.	2 wks.	22	450 cc.
13	45	M.	Endocarditis	(?)	....	1 mo.	25	500 cc.
14	19	M.	Endocarditis	Rheumatic fever	10 mos.	Continued	20	350 cc.
15	43	M.	Myocardial degeneration	Hypertension; arteriosclerosis	9 yrs.	4 mos.	22	550 cc.
				Thirteen Days Later				
16	36	M.	Endocarditis	Rheumatic fever	....	1 wk.	15	550 cc. 265 cc. (for pulmonary infarction)
17	65	M.	Endocarditis	Rheumatic fever	6 wks.	Continued	Edema	600 cc.
18	52	M.	Myocardial degeneration	Arteriosclerosis	....	Continued	20–22	500 cc. (convulsion with apoplexy)
19	51	M.	Myocardial degeneration	Hypertension; arteriosclerosis	6 mos.	1 wk.	18	350 cc.
				Four Days Later			24–26	400 cc.
20	75	M.	Myocardial degeneration	Arteriosclerosis	6 yrs.	2 wks.	24	500 cc.
21	50	M.	Myocardial degeneration	Arteriosclerosis	11 mos.	Continued	20	400 cc.

TABLE III

Clinical Data on Venesected Cases

Resultant V. P.			*Effect*	
Lowest	*Interval*	*Duration*	*Immediate*	*Ultimate*
14	30 min.	Maintained	Little change early	2 days later less cyanotic, less edema, diuresis
15	24 hrs.	Maintained	Immediate improvement mental state and objectively	Death 5 months later, V. P. 13.
10	24 hrs.	10 days later 16 cm.	Dyspnea lessened, output increased	Slump on 11th day, death on 33d day
12	Immediate	5 hrs. later 16 cm. 24 hrs. later 19 cm.	Immediate subjective improvement in breathing, cyanosis same	
			Three Days Later	
14	Immediate	24 hrs. later 16 cm.	Early relief dyspnea cyanosis	Died 36 hrs. after venesection
Edema	.....	.....	No	Died 2 days later
13	10 min.	1 hr. later 15 cm., 24 hrs. later 23 cm.	No response	Died 20 days later
15	48 hrs.	3 days later 24 cm.	No response	Died 10 days later
14	Immediate	Irregular from Cheyne-Stokes, 13 –20 cm. Steady fall	Less abdominal distress; improving subjectively	Remarkable return of circulatory balance; discharged 6 wks. later V. P. 10 cm.
11	Immediate	5 hrs. later 14 cm.	Marked subjective improvement	Progressive improvement; discharged 7 weeks
8	1 hr.	Not above 12 cm. until death	Less restless; less cough; less pulmonary congestion	Infarction determined death 11 days later
Edema	.....	.....	Pronounced subjective and objective improvement beginning in 24 hrs.	Death 6 wks. later from cerebral hemorrhage
11	Immediate	.....	Slight but very temporary improvement	Death 27 hrs. later from pulmonary infarction
12	48 hrs.	11–15 cm. until discharged	Marked subjective improvement	Discharged markedly improved 7 wks. later
17	24 hrs.	Steady fall to 13 cm. in 10 days	Marked subjective improvement	Death 12 days later
12	24 hrs.	5 days later 16 cm.	No improvement	
			Thirteen Days Later	
.....	.....	.....	No response	Death 2 days later
12	30 min.	Fall to 7 cm. in 24 hrs.	Pronounced improvement in general condition, less cough	V. P. remained normal until discharge 6 wks. later
Edema	.....	.....	Marked subjective improvement	V. P. 10 cm. 11 days later; steady gain to discharge 5 wks. later
13	3½ hrs.	Slight rise but fall to 12 in 24 hrs.	Apparent early improvement; later disappearance of cyanosis	Sudden death 2½ wks. later
16	Immediate	No real gain, 24 cm. 4 days later	No response	
			Four Days Later	
16	Immediate	12 hrs.	Definitely less dyspneic	Died 31 hrs. later
11	2½ hrs.	24 hrs. later 21 cm.	No response	Died 2 days later
5	Immediate	1½ hrs., 2 hrs. later 8 cm.	Respiration less labored; fewer râles	Sudden collapse and death 8 days later

degree explain the high mortality in spite of any form of treatment.

The immediate effect of venesection is, as we have seen, purely mechanical, and serves merely to reduce the high load to which the right heart is exposed as a result of the venous engorgement and high venous pressure. It is a load which is higher than the range within which the muscle can respond efficiently or adequately. This range has been reduced as a result of disease and the unfavorable situation that exists in decompensation, below the normal. It is always a question whether, even by considerable reduction of the venous blood volume, the load can be brought within this narrowed range. If so, an immediate response may be expected, and even a brief respite may go far to assist the heart in attaining a greater range of adequate response. Lasting and perhaps permanent improvement with complete restoration of compensation may result. On the other hand, if the range of normal response has been too much narrowed by disease or by the severity of the decompensation, the load may not be reduced sufficiently to come within this range, or, if so, the improvement in the muscle may not be great enough to maintain an adequate response. The fall of venous pressure is temporary, and it quickly returns to, or even above, its original level. Experience has shown that if the response to a single venesection is inadequate, little can be expected from subsequent efforts in this direction. An initial

favorable response may be again obtained, but is not permanent in those instances where an earlier effort has been unsuccessful. The most accurate index to the response to venesection is the venous pressure. If it falls following the procedure and remains low, the symptoms and signs of congestive failure subside rapidly. If the pressure returns rapidly following the initial fall, only a temporary improvement can be expected. The degree and especially the permanency of the response depends upon the recovery ability, and the extent of damage in the cardiac muscle. The most reliable guide to the clinical indication for venesection, and to the character of response that is obtained, is the frequent determination of venous pressure. Here again it is the trend of the pressure, rather than the actual height, which has the greatest significance.

Recent work (61) has tended to emphasize the damage done to cardiac muscle by the abnormal stretching that occurs when it is exposed to an overload, and the resulting unfavorable after effects of this initial injury. The probability of an early recurrence of decompensation subsequent to the first period of failure is well recognized clinically. It is beginning to be apparent that this is largely due to the additional injury suffered by the cardiac muscle during the initial period of failure, and that this injury has, as its underlying basis, a mechanical factor. It appears, therefore, of essential importance to protect the muscle, so far as possible, even

in relatively mild failure, against this factor, and in addition to rigidly enforced rest, venesection is certainly the most effective form of therapy. In the opinion of the writer, it is a procedure which should be carried out early in many cases of decompensation and not reserved, as is usually the case, until other measures of treatment have failed. Not only is it often an essential method in the rapid restoration of cardiac competence, but the muscle is protected against prolonged overload with consequent injury from the mechanical stretching, as well as from a long period of malnutrition and anoxemia.

It is difficult to separate the influence of drug therapy in cardiac decompensation and the effect upon venous pressure from that of rest and other measures. No drug is known which in the normal animal has a specific effect in lowering venous pressure (99). The members of the digitalis series may be said more nearly to approach this through their influence in increasing cardiac activity, although the trend of recent work would indicate that this effect is confined to the conditions existing in cardiac decompensation (100) (101) (102), and that in the normal subject no such effect, or perhaps the opposite influence prevails. The very marked influence of digitalis in congestive failure, particularly when associated with auricular fibrillation, has long been evident. The presence of a rapid auricular fibrillation results in a condition which, from the mechani-

cal standpoint alone, greatly reduces the efficiency of the heart as a pump (59) (103). In this instance, the influence of digitalis is in reducing conductivity over the auriculo-ventricular conductive system, and in protecting the ventricle from the bombardment of supraventricular impulses, and thus insuring a slower, more regular, and efficient contraction. As a result, there is an increase in minute volume output, and a more effective emptying of the venous system. This leads in favorable cases to a fall of venous pressure, reduction of venous and capillary engorgement, reëstablishment of renal function, and rapid disappearance of the signs and symptoms of vascular congestion. Its influence is most pronounced, as noted first by Withering (104), in those cases in which "the pulse be feeble or intermittent, the countenance pale, the lips livid, the skin cold, the swollen belly soft and fluctuating, or the anasarcous limbs readily pitting under the pressure of the finger, and we may expect the diuretic effect to follow in a kindly manner." Modern work is almost uniform in ascribing the beneficial effects of digitalis, including diuresis, to its action on the decompensated heart (105) (106) (107) (108), and through this in reducing vascular congestion. Its effect upon the heart may be in large part due to a reduction of the anoxemia, associated with the slower rate of contraction (24) (25) (29). It is obvious that the effect of digitalis upon venous pressure, while indirect, may be profound in those types

of failure where the influence of digitalis upon the heart leads to improvement in its mechanical efficiency. A rapid fall of venous pressure following the injection of oubain intravenously has been observed (96).

Diuretics, other than digitalis, may be presumed to contribute to the lowering of venous pressure in the restoration from congestive heart failure when effective in causing diuresis. The reduction of peripheral resistance in previously water-logged tissues and the consequent reduction of load upon the left heart, and the decrease in total blood volume that probably results, would be factors assisting in the return of normal heart function. So far as I know there are no direct observations of venous pressure under the influence of diuretics, other than digitalis, when exhibited alone in cardiac failure, but recent work has tended to emphasize the fact that they may be effective in producing diuresis in cases in which digitalis has failed. Marvin (109), in a study of seventy-seven cases of congestive heart failure, observed for three weeks or longer, found that thirty-six, or 47 per cent, were rendered edema free by rest, or by rest and digitalis alone. Of the remaining forty-one, or 53 per cent, thirteen were completely relieved of their edema by the xanthine diuretics, while in the remainder varying degrees of diuresis were produced, without, however, entire removal of the excess fluid. The most potent of the diuretics was theocin, effective in a number of

cases in which theobromine or theobromine sodio-salicylate had failed. Eleven of the thirteen patients responding to diuretics other than digitalis belonged to the arteriosclerotic-hypertensive group, while only two of the eighteen patients belonging to the rheumatic group gave a satisfactory response. Van Valzah, McKinley, and Middleton (76) attribute an even higher value to novarsurol, and also regard the Niemeyer pill (quinine 60 mgs., digitalis 5 mgs., opium 15 mgs.) as worthy of serious consideration.

Cardiac failure in lobar pneumonia. It is well known that in a certain percentage of cases of lobar pneumonia cardiac incompetence develops, and it is regarded as a frequent cause of death in this disease. The development of incompetence in lobar pneumonia would appear to be associated with two factors: first, an increase in resistance in the pulmonary circuit due to the pathological changes in the lungs and leading to an overload upon the right heart, and secondly, to a reduction of cardiac muscle efficiency as a result of the toxemia and perhaps the anoxemia. Massive involvement of the lungs may occur without evidence of cardiac incompetence, while this condition may be manifest in less extensive anatomical involvement when evidences of toxemia are pronounced. The two factors necessary for cardiac decompensation in primary organic heart disease, namely, increased heart load, and a lowered range of response on the part of the cardiac muscle, thus appear also as the two necessary factors in the

heart failure of lobar pneumonia. In the former case, the reduced range of physiological response is due to chronic disease of the muscle, associated, probably, with pathological changes incident to, and coincident with, hypertrophy. On the other hand, in pneumonia it appears to be in the nature of toxic myocarditis, or a local manifestation of a toxemia which may affect other organs as well. In many cases of lobar pneumonia with apparently normal cardiac function, there appears to be a slight degree of cardiac dilatation (110), possibly due to the effect of the toxins associated with this disease.

The venous pressure in lobar pneumonia may be described in a few words. It is normal so long as the heart is sufficient and a normal circulation is maintained (40) (110) (2), while it shows the usual rise characteristic of cardiac decompensation if this condition develops. From the standpoint of dynamics of the circulation, cardiac failure in lobar pneumonia does not appear to differ in any essential details from the same condition developing in primary heart disease. It is associated with engorgement and a sluggish flow in the capillaries (2), a definite rise of venous pressure, and the usual signs and symptoms of cardiac failure. Restoration of compensation is accompanied by a falling venous pressure, a return to normal flow and extent of filling of the capillaries, and a disappearence of the signs and symptoms of an insufficient circulation. Venous pressure observations in every case of lobar

pneumonia would appear to be an essential practice in reference to the information it supplies as to the status of the cardio-vascular system in this disease. A rising venous pressure may be the first indication of the impending onset of other signs and symptoms of cardiac incompetence, and the progress of the incompetence, when once established, can be more accurately judged from the trend of the venous pressure change than by any other method (Case VI, Chapter VII). As a guide to venesection and other methods of therapy in this complication of pneumonia, it is of significant value.

CHAPTER VI

Venous Pressure in Conditions Other Than Cardiac Failure

Chronic heart disease. According to most observations, venous pressure is normal in chronic heart disease so long as cardiac compensation is well maintained (40) (53) (36) (41) (44) (2). An exception has been made by some observers of aortic regurgitation (42), denied, however, by others (41) (44). In cases of chronic heart disease where compensation is imperfect and yet fairly well maintained, venous pressure may be in the upper range of normal (80-110 mms.), and above the average normal (60 mms.) (2) (44).

Paroxysmal tachycardia and fibrillation. Cases of paroxysmal tachycardia in which the venous pressure rose to abnormal heights have been recorded (32) (53). In these cases there were associated signs of venous stasis (cyanosis, dyspnea, liver engorgement, albuminuria), so they are really to be regarded as cases of temporary cardiac incompetence resulting from the very high ventricular rate. The ventricles were beating so rapidly that the period of diastolic filling was interfered with,

and, in spite of the high rate of contraction, or rather due to this, the volume of blood pumped in a unit of time was reduced (111).

In paroxysmal fibrillation unassociated with signs of venous stasis, venous pressure is little altered (2). It would seem, therefore, that venous pressure may or may not rise in paroxysmal tachycardia or fibrillation, depending upon whether or not cardiac incompetence supervenes.

Miscellaneous conditions involving the cardiovascular system. In acute and subacute endocarditis, venous pressure is normal unless cardiac failure supervenes (2) (112). In arterio-venous aneurism, the general venous pressure is somewhat increased, but is usually higher in veins draining the region where the anastomosis occurs than in those from other regions (113) (112). Dry pericarditis is associated with a normal peripheral venous pressure (112), but observations appear to be lacking in cases of pericarditis with effusion when unassociated with signs of cardiac failure. Pernicious anemia, although associated with marked changes in cardiac output and blood flow (114), exhibits normal venous pressure (2) (36).

Peripheral vasodilatation. The fact that venous pressure is relatively independent of the degree of filling and pressure in the small vessels of the peripheral circuit has already been discussed. A tendency to slight venous hypertension in a series of cases classified as vasomotor instability has been

described by Kroetz (53). This finding has not, however, been substantiated by other workers (2).

Arterial hypertension. The question as to whether venous pressure is elevated in well-compensated cases of arterial hypertension has been much discussed. From the experimental standpoint, it has been shown by various workers that alterations in arterial pressure affect venous pressure little if at all, unless cardiac activity is simultaneously affected. Some observers, however, have found in man a definite relation between the two pressures, both in the normal and in arterial hypertension, the venous pressure being maintained at about one-thirteenth of that of the pressure in the larger arteries (42). The majority of workers find no such relationship, and the preponderance of evidence points to the conclusion that venous pressure is high in cases of arterial hypertension only in the presence of cardiac incompetence (41) (40) (44) (115) (116). The accompanying table gives the results of readings in eighteen cases of hypertension in the wards of the Wisconsin General Hospital. It would appear from this that in certain types of arterial hypertension, at least, the venous pressure is in the upper range of normal.

Pulmonary conditions other than pneumonia. The possible influence of various abnormal states in the lungs and thorax, in modifying systemic venous pressure through the effects they may exert on intrathoracic pressure, has been emphasized by

TABLE IV

VENOUS PRESSURE IN ARTERIAL HYPERTENSION

Groups	*Number of Cases*	*Arterial Pressure in mms. of Mercury*		*Venous Pressure in Recumbent Position. In mms. of Water*	
		Averages			
		Systolic	*Diastolic*	*Average*	*Highest*
Chronic nephritis and hypertension	9	196	128	60	80
Hypertension with arterio-sclerosis..	4	217	143	88	110
Hypertension without arterio-sclerosis.....	5	203	116	90	120
General average...	18	205	129	79	

Kroetz (53). Conditions altering the physical situation in the thorax, such as empyema, pneumothorax or pronounced scoliosis, or in the lungs, such as emphysema and pleural adhesions, may, according to this observer, so alter the venous pressure that when read in a peripheral vein it may be no index of the "effective venous pressure" (right auricular pressure) at the heart. In two cases in which intentional pneumothorax was produced, this procedure resulted in a change of peripheral venous pressure, but there was reason to believe that the venous pressure in the heart was unchanged or less altered. Kroetz concluded that in certain cases of abnormal thoracic or pulmonary states associated

with cardiac decompensation, peripheral venous pressure may be within the normal range, while the "effective venous pressure" is high. He found an abnormally high venous pressure in all cases of decompensation when the lungs and thorax were approximately normal. In eight cases of advanced kyphosis and scoliosis, and in one case of empyema in the wards of the Wisconsin General Hospital, normal venous pressures were found in the recumbent position. The average reading was 50 millimeters, the highest 80 millimeters of water. It should be noted in this connection that it has been shown experimentally that the injection of fluid into the thorax causes no rise of peripheral venous pressure until the period immediately preceding death, at a time when asphyxia has resulted from compression of the lungs (81).

In pulmonary tuberculosis, an elevation of venous pressure has been recorded in advanced cases (34), or where arterial hypertension is present (40). Others have found the pressure normal in phthisis except when heart failure supervenes (44), or have distinguished between different types of the disease; a rise occurring only in the miliary and fibroid types (115).

In emphysema varying results have been recorded. In one advanced case Gaertner (34) recorded a venous pressure reading of 180 mms., but failed to note whether there were accompanying signs of cardiac incompetence. Others have found a rise only

when acute pulmonary edema occurs (96) (118), ascribed not to a primary heart incompetence, but to rise of tension in the pulmonary circuit. In eleven cases of emphysema in the wards of the Wisconsin General Hospital the highest venous pressure was 90 millimeters, and the average reading for the group was 68 millimeters of water. Finally, venous pressure has been stated to be usually below the average in emphysema (53).

It is important to note that with few exceptions (34) these divergent results concerning venous pressure in states other than cardiac decompensation have come from observers employing the direct method of cannulation of the vein, a method which there is abundant reason to believe may readily lead to erroneous and to divergent results in the hands of different observers. It is also not always evident that posture and body rest have been controlled in these observations.

High venous pressure in mediastinal growths and in struma, due to direct pressure on the veins entering the heart, have been described by several observers (56) (53) (2). In another case of pulmonary neoplasm, normal venous pressure was recorded (112), presumably because pressure and stasis was not present.

Local venous obstruction. Extremely high venous pressures up to 1620 mms. of water (120 mms. of mercury) are said to occur from direct compression of the veins of a limb with the resulting

interference with venous return (119). Occasional high readings of peripheral venous pressure in apparently normal individuals have been referred to compression of the peripheral veins by excessive muscular development (53) or obesity (58). Local obstruction due to mediastinal growths has been mentioned. In a reported case of Hodgkin's disease with extensive mediastinal involvement, the venous pressure in the veins of the hand rose gradually from 80 to 220 mms. as the condition progressed (2). Venous pressure in the veins of the lower extremities was normal throughout. Autopsy revealed partial occlusion of the superior vena cava. A rise of pressure in the leg veins during pregnancy (46), and in varicose veins when the condition is the result of obstruction (120) (41), have been noted. In varicosities not due to obstruction, venous pressure was low in the affected veins in the prone position, but rose to exceedingly high values (990 mms. of water) in the erect position, ascribed to the effect of gravity upon vessels in which the normal tonus had been lost. Middleton (121) reported a case of aortic aneurism in which venous pressure was approximately twice as high in the veins of the left hand as in those of the right, presumably due to obstruction to venous return. The exceedingly high venous pressure of 490 mms. of water in a case of polyserositis involving the pleura, pericardium, and peritoneum, and due presumably to stasis from obstruction to venous return, has been reported by Ayerza

(112). Phlebosclerosis has been mentioned as the occasional cause of high venous pressure readings (32), but it appears to be quite rare (2).

Miscellaneous disease. The highest venous pressure observed by Gaertner (34) (1300 mms. of water) was in a case of cachexia with infiltration at the apices of the lung. The cause of the rise, or whether it was associated with signs of decompensation, is not clear. Moreover, von Basch (31), Moritz and Tabora (37), and Eyster and Middleton (2) have failed to find any noteworthy elevation of venous pressure in various diseases not involving directly or indirectly the cardiovascular system. A new clinical syndrome, showing as its main features cyanosis, venous hypertension, and evidences of ovarian insufficiency, and occurring principally in young women, has been recently described (122).

Venous pressure in general anesthesia. Observations during general anesthesia made by Middleton and Meyer show an invariable rise of venous pressure during the stage of excitement, falling toward normal as full surgical anesthesia comes on. In twenty-seven cases, the average venous pressure before anesthesia was 70 mms. of water, the highest 90 mms. The average pressure recorded during anesthesia was 209 mms.; the highest, 270 mms.; and the lowest 120 mms. A rise occurred in all cases. Not much difference appears to be present with the different anesthetics used. The

average highest pressure with nitrous oxide was 168 mms.; with ethylene, 175 mms.; and with ether, 230 mms. The rises in each case appear to be associated with the periods of excitement and struggling. These observations obviously point to the importance of avoiding these features of anesthesia in so far as possible, when a general anesthetic is necessary in a subject with organic heart disease.

CHAPTER VII

Clinical Cases

Cardiac decompensation supervenes, as is well known, in two types of chronic heart disease, classified according to their etiology into the rheumatic or endocarditic group, and the myocardiac or arteriosclerotic group. In a series of 36 cases of congestive heart failure, selected from the records of the wards of the Wisconsin General Hospital, the following general points are of interest.

Endocarditic group. This group includes 17 cases, 12 of which were in women. The average age of the patients was 41 years; the oldest, 62; and the youngest 23. Nine, or 53 per cent, died in the hospital. There was a previous history of cardiac decompensation in 11, or 65 per cent. The average period preceding the present decompensation when symptoms were first noted was 42 months. The shortest period was one month, the longest 108 months. The average venous pressure on admission was 200 mms. of water; the lowest, 140 mms.; and the highest, 280 mms. There was no important difference in this respect between the fatal and the recovery groups. The average venous pressure shortly before death in the fatal cases averaged 190

mms.; the lowest, 110 mms.; the highest, 290 mms. In the 8 cases recovering the average venous pressure on discharge was 70 mms.; the lowest 50 mms.; and the highest, 90 mms.

Myocardiac group. This group includes 19 cases, only 3 of which were in women. The average age of the patients was 59½ years; the oldest, 85; and the youngest, 41. Seven, or 37 per cent, died in the hospital. There was a previous history of decompensation in 9, or 47 per cent. The average period preceding the present decompensation when symptoms were first noted was 17½ months, the shortest period was 2 weeks, the longest, 72 months. The average venous pressure on admission was 180 mms. of water; the lowest, 140 mms.; and the highest, 280 mms. This average was actually a little higher in the cases recovering than in those dying; 190 and 170 mms. of water, respectively. The average venous pressure shortly before death in the fatal cases was 180 mms.; the lowest, 140 mms.; and the highest, 230 mms. In the 12 cases recovering, the average venous pressure on discharge was 80 mms.; the lowest, 60 mms.; and the highest, 110 mms.

A comparison of these two groups brings out the general clinical differences usually noted; the difference of the age incidence of cardiac failure, the tendency for a longer period of symptoms preceding the actual decompensation, and the more frequent occurrence of repeated decompensations in the endocardial group. There are no striking differences in

the venous pressure findings between the two groups. It appears to be slightly higher on the average in the endocarditic group, both on admission, and before death in the fatal cases. In neither group is the venous pressure higher on entrance in the fatal cases than in the recovery cases. This serves to emphasize that it is not the actual height of venous pressure which is of major importance, but the trend under treatment. The maintenance of a high venous pressure in the cases proceeding to a fatal termination, as well as the fall to within the normal range of the cases in both groups in which compensation was restored, is obvious from the data.

The following cases are presented to bring out certain details in reference to the venous pressure findings. The first case shows two well-defined periods of decompensation, each responding favorably to treatment.

Case I.—Mrs. A. F. S.—age 47. Health was good until nine years ago when there was a transient hemiplegia, apparently precipitated by exertion and nervous strain and lasting less than a day. Patient felt well until a year ago, although she had noticed a tendency to fatigue and some shortness of breath on exertion. While on a fishing trip, a severe chill occurred, associated with palpitation and orthopnea. Irregularity of heart beat was noted at this time, and has persisted until the present. The succeeding five weeks were spent at bed rest, but improvement was slow. Cough developed, and there was

some shortness of breath even when at rest. Ankle edema was noted four months ago, and this was followed by abdominal distension and spreading of edema to the legs. The patient gives a history of scarlet fever and rheumatic fever at ten years of age, another severe attack of rheumatism at sixteen years, and a third attack at twenty-one years. An attack of chorea occurred at sixteen. The history also includes diphtheria at eleven and pneumonia at eighteen. There is no history of pregnancies or miscarriages. Patient is in obvious discomfort and quite dyspneic.

Physical examination reveals moderate cyanosis of face and mucous membranes, chest of emphysematous type with signs of fluid at right base and diffuse edema of lungs of moderate grade. The heart impulse is diffuse in the fifth and sixth interspaces extending beyond the midclavicular line. The rhythm is totally irregular, and there is a loud blowing systolic murmur, maximum at the apex, and transmitted to the posterior axilla. The pulmonic second sound is accentuated. The abdomen is markedly distended, the walls are edematous, and there is movable dullness in the flanks with characteristic succussion waves. The liver and spleen are enlarged. There is moderate cyanosis and edema of arms and legs. Systolic arterial pressure is 140 mms. of mercury; venous pressure, 220 mms. of water (Fig. 3). The pulse deficit is 14, with a cardiac rate of 124. The urine showed a high specific gravity and a small

amount of albumin. The blood showed slight secondary anemia. The electrocardiogram revealed auricular fibrillation with periods of flutter, right ventricular preponderance and inversion of T_3.

The diagnosis was mitral valvulitis, stenosis and insufficiency, cardiac hypertrophy, auricular fibrillation, cardiac decompensation, pulmonary edema, ascites, hydrothorax, anasarca, and chronic passive

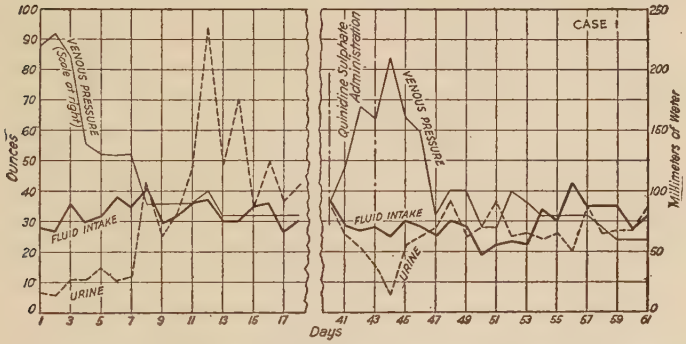


Fig. 3.—Chart showing the venous pressure in relation to the fluid intake and urine output. Case I.

congestion of liver and spleen. Treatment consisted of bed rest, milk diet with total fluid intake restricted to 40 ounces daily, digitalization, and magnesium sulphate each morning. The venous pressure began to fall by the third day, and shortly after that a definite diuresis occurred, associated with marked clinical improvement and disappearance of the edema. By the fourteenth day the peripheral edema had nearly disappeared, and the free fluid in chest and abdomen was much reduced.

The diuresis continued to the twentieth day, when a positive water balance was restored. The venous pressure was within the normal range subsequent to the seventh day. On the thirtieth day all evidences of edema and free fluid had disappeared. The systolic arterial pressure was 116; diastolic, 76; venous pressure, 80 mms.; apical rate 72 with no pulse deficit; and patient free from dyspnea or other discomfort. The condition continued favorable to the fortieth day, preceding which for ten days the patient had been in a chair for increasing periods daily. On the fortieth day a course of quinidine sulphate medication was begun in an effort to restore a normal cardiac rhythm. The drug was administered in doses of 0.25 grams every fourth hour for four doses. After three doses, auricular tachycardia developed with a rate of 180, and a short time later periods of auricular fibrillation alternating with periods of rapid regular heart action were noted and recorded electrocardiagraphically. Palpitation, dyspnea, and cyanosis developed, associated with a rise of venous pressure from 90 to 120 mms. Urine output fell from 36 to 28 ounces (1080 cc. to 840 cc.). During the night, a few hours after the last dose of quinidine for that day, heart action was more stable, and patient was in less discomfort. On the following day quinidine administration was carried out as on the day preceding. Palpitation, cyanosis, and dyspnea again appeared; the venous pressure rose sharply, and the urine output declined. Elec-

trocardiagrams showed periods of auricular fibrillation interspersed with periods of rapid regular heart action. The symptoms subsided somewhat with the cessation of quinidine administration that night. The following morning quinidine administration with the same dosage was begun again. A short time after the first dose extreme distress developed. There was dyspnea (respiratory rate 36), marked cyanosis, palpitation, pain in side, and cough with expectoration of blood-streaked sputum. Examination revealed right-sided hydrothorax, pulmonary edema, ascites, tympanites, and slight edema of the extremities. Venous pressure rose to 18 cm. Electrocardiagrams revealed the same type of rhythm as on the two previous days. The cardiac area was increased (teleroentgenogram). Due to the critical condition of the patient, and the absence of any favorable action on the cardiac rhythm, quinidine administration was stopped after the third dose on this day, and digitalization with general treatment for cardiac decompensation established. A total of 2.75 gm. quinidine sulphate had been administered. On the following two days the condition was critical. The venous pressure had risen to 21 cm.; the urine had fallen to six ounces (180 cc.) in twenty-four hours. Continuous auricular fibrillation persisted with an average apical rate of 136, and radial of 82. By the next day (May 22) the general condition had begun to improve. Venous pressure had fallen to 16 cm. and urine increased to 16 ounces (480 cc.).

Steady improvement continued, and on June 3 the patient was discharged from the hospital, free from any symptoms or signs of decompensation. Auricular fibrillation, however, was still present, with an apical rate of 72, and with a very small pulse deficit. On discharge, the venous pressure averaged 7 cm., and the urine output was approximately equal to the fluid intake.

Case II. The second case shows a tremendous diuresis with favorable response to combined therapy, as occurs in certain cases of congestive heart failure.

L. E. H., male, age 76, entered hospital complaining of swelling of legs and shortness of breath. Two years ago he had a transitory left-sided paralysis followed by muscular weakness. Shortness of breath had been noticed for the last three months, especially bad at night, and becoming progressively worse. He suffered also from vertigo, double vision, palpitation and precordial pain, cough, and swelling and numbness in arms. The past history is negative except for childhood diseases. This patient had been living alone for the past five years in the woods in northern Wisconsin. He was found ill and helpless by the representative of a humane society of a neighboring town, and sent to the hospital.

Physical examination revealed marked dyspnea, cyanosis of skin and mucous membranes, edema of eyelids and face and general anasarca. There were signs of fluid in the right thorax below the seventh

rib, and scattered fine and coarse moist râles over the remainder of the lungs, with the physical findings of emphysema. The heart was enlarged to the left and totally irregular in force and rhythm. The apical rate was 95, radial 81. Systolic arterial pres-

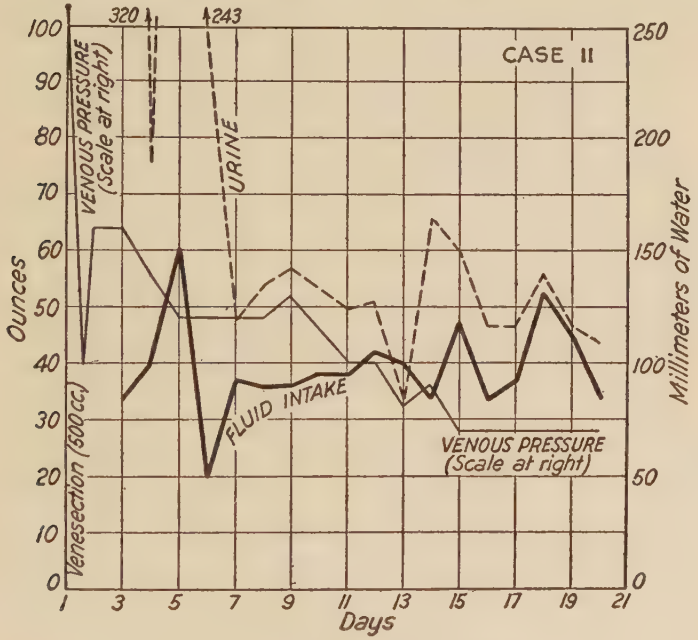


Fig. 4.—Case II. Similar to Fig. 3.

sure was 155 mms. of mercury; the venous pressure, 260 mms. of water (Fig. 4). The abdominal walls were edematous, and there were signs of a moderate amount of free fluid. The liver edge was felt 8 cms. below the costal margin in the midclavicular line. The spleen was not palpated. Urine showed a

trace of albumin. The non-protein nitrogen of the blood was normal, and the blood Wassermann was negative. The phthalien excretion was reduced to 15 per cent in two hours. The electrocardiagram revealed auricular fibrillation, left ventricular preponderance, intraventricular conduction time increased, T_1 negative, and T_2 flat. An x-ray film of the chest showed complete clouding at the base of the right lung. The diagnosis was chronic myocarditis with hypertrophy and dilatation, cardiac decompensation with anasarca, hydrothorax and ascites, chronic passive congestion of the liver, and auricular fibrillation.

Treatment consisted of confinement to bed with back rest, and milk diet with fluid limitation to 1800 cc., and morphine. Venesection was done the afternoon of the admission day, with removal of 500 cc. The following day digitalization was started and coramine was also administered. The venous pressure fell to 100 mms. following the vensection, with a rebound to 160 mms. the following morning, and a steady fall toward normal after the third day. The urine output rose to 320 ounces (9900 cc.) on the third day, and to 243 ounces (7560 cc.) on the fifth day. With this tremendous diuresis there was evident decrease in edema and marked clinical improvement. On the second day 1000 cc. of fluid was removed by thoracentesis. The progress toward restoration of compensation proceeded uneventfully. Venous pressure was within the normal range after the tenth day. The patient was discharged without

evidences of edema or cardiac insufficiency. The auricular fibrillation persisted.

Case III. The third case illustrates an early favorable response with a fall of venous pressure, diuresis, and clinical improvement, with subsequent rise of venous pressure, and the establishment of a variable and unsatisfactory venous pressure level and water balance, with gradual water retention. The clinical condition became progressively worse, and death occurred on the sixtieth day in the hos-

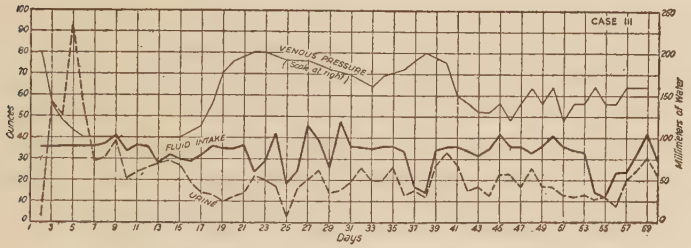


Fig. 5.—Case III. Similar to Fig. 3.

pital. The general tendency for an inverse relation between venous pressure and urine output throughout the clinical course is well illustrated in this patient (Fig. 5).

L. J., female, age 62, entered the hospital complaining of shortness of breath. This had been present for the past six months and had been much worse recently. There had been slight swelling of the ankles and gas eructations after eating. The past medical history was negative except for repeated attacks of severe tonsillitis until removal of tonsils

ten years ago, and diphtheria. Social and family history were unimportant.

On physical examination there was marked dyspnea and orthopnea, cyanosis and distension of superficial veins. The thorax showed bulging over the pericardium. There was inspiratory retraction of lower left interspaces and the physical findings of marked pulmonary congestion. The heart apex was visible in the seventh intercostal space in the anterior axillary line. There was a presystolic thrill and murmur at apex, and a loud systolic murmur, maximum at the apex, and transmitted to the left posterior axilla and infrascapular space. A systolic murmur of different quality was heard at the base of the heart and transmitted to the vessels of the neck. Arterial blood pressure: systolic, 156; diastolic, 104 mm. of mercury. Venous pressure 200 mms. water. The abdomen was distended with movable dullness in both flanks. The liver was palpated at the level of the umbilicus, and was tender, but did not appear to pulsate. Spleen was not felt. The urine contained moderate amounts of albumin and a few hyalin casts. The blood showed a slight grade of secondary anemia. The non-protein nitrogen of the blood was normal, and blood Wassermann was negative.

The diagnosis was mitral stenosis and regurgitation, aortitis, cardiac hypertrophy and dilatation, chronic passive congestion of lungs and liver, and ascites.

Treatment consisted of rest in bed, milk diet, with total fluid intake limited to 1200 cc., digitalization, and morphine for relief of severe attacks of orthopnea. A prompt fall of venous pressure, the establishment of diuresis, and definite clinical improvement occurred. The venous pressure had fallen to within the upper normal limits by the fifth day, when the urine secretion reached its maximum. The signs of chronic passive congestion in the lungs were much reduced. The liver became smaller, and the ascites less evident. Improvement continued until the seventeenth day, when nausea developed. This was associated with a beginning rise of venous pressure, a fall in urine secretion, and on the following day the liver enlargement, ascites, and abdominal distention had definitely increased. The patient's general condition became definitely worse. Within a few days venous pressure had again risen to 200 mms. of water, and urine secretion had dropped to a very low point. Signs of pulmonary congestion also increased. Subsequent to this, the venous pressure fell gradually, and urine secretion rose with subjective improvement and some decrease of signs of chronic passive congestion and ascites. The fall of venous pressure was slow, however, and the lowest point recorded was 160 mms. Subsequent to the thirty-third day, venous pressure started a gradual rise associated with the development of another low point in urine output, with increase in signs of congestion, and increased cyanosis, dyspnea, and weak-

ness. Jaundice and edema of lower extremities appeared. Venous pressure fell somewhat following the thirty-seventh day, but was quite variable and well above normal. Urine output remained low, the patient became progressively weak, with increase of congestion and edema, and died on the sixtieth day after admission. Autopsy revealed chronic mitral endocarditis with stenosis and regurgitation, cardiac hypertrophy and dilatation, arteriosclerosis, chronic myocarditis, infarction of lungs, chronic passive congestion of lungs and abdominal viscera, and chronic nephritis.

Case IV. This case illustrates a partially favorable venous pressure response to combined therapy, with slow establishment of moderate diuresis, and the final establishment of a normal venous pressure level and water balance, with restoration of decompensation after a rather prolonged period.

W. J. B., male, age 69, had been admitted to the hospital two years previously, following transitory left-sided paralysis and mental confusion. Diagnosis at that time was cerebral and general arteriosclerosis, arterial hypertension (systolic pressure 188, diastolic 110 mms. of mercury), myocarditis, and mitral regurgitation (relative). The patient was discharged after three days in good condition. On reëntrance to the hospital, the principal complaint was shortness of breath, which had begun about two months before, and was becoming progressively worse. He complained also of swelling of ankles and

abdominal distress and distention. He had been taking digitalis for ten days before entering the hospital.

Physical examination revealed normal mental response, moderate dyspnea, slight cyanosis of mucous membranes, and edema of ankles and legs. The lungs showed signs of moderate congestion, but there

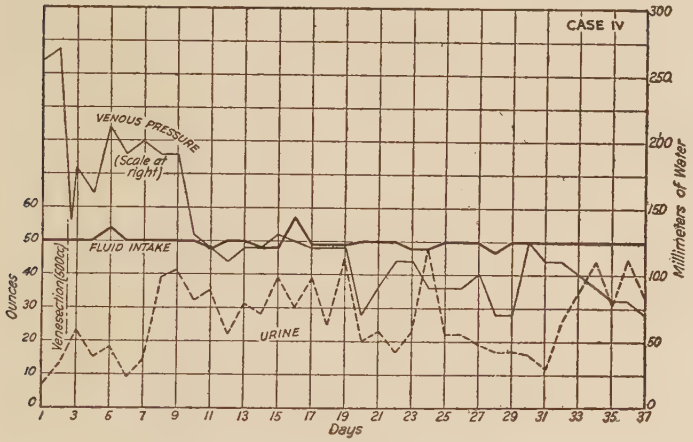


Fig. 6.—Case IV. Similar to Fig. 3.

was no evidence of free fluid in the thorax. The heart impulse was in the fifth interspace, and was visible as far out as the anterior axillary line. There was a loud, blowing systolic murmur transmitted to the axilla. The arterial systolic pressure was 172 mms.; the diastolic, 116 mms. of mercury. Venous pressure was 260 mms. of water (Fig. 6). The abdomen showed no evidence of free fluid. The liver edge was palpated 10 cm. below the costal margin in the

midclavicular line. The spleen was not palpated. The urine showed a high specific gravity, moderate albumin, and many hyaline casts. The blood showed a slight grade of secondary anemia. The non-protein nitrogen of the blood was normal. The blood Wassermann was negative. Phthalein urinary output was normal. The electrocardiogram revealed left ventricular preponderance, T_1 and T_2 diphasic, and prolonged auriculo-ventricular conduction time (0.22 sec.).

The diagnosis was chronic myocarditis, cardiac hypertrophy and dilatation, generalized arteriosclerosis, arterial hypertension, cardiac decompensation, mitral regurgitation (relative), chronic passive congestion of lungs and liver, and anasarca.

Treatment consisted of bed rest, low protein salt-free diet with limitation of fluid intake to 1500 cc., digitalis, and morphine. Venesection was done on the first day with withdrawal of 500 cc. Venous pressure fell to 140 mms., but rebounded shortly to 180 mms. The urine secretion increased, but fell again as the venous pressure rose to 210 mms. Niemeyer pills (one) were then administered twice daily, and on the sixth day theobromine sodium salicylate was started in doses of five grains three times daily. Urine output rose following the seventh day, and venous pressure dropped quite sharply to slightly above the upper normal range. There was no improvement in signs or symptoms until this occurred, when clinical improvement and decrease

in signs of congestion and edema proceeded gradually but uninterruptedly. Compensation was gradually restored, a satisfactory water balance was obtained, venous pressure fell to within normal limits, and the patient was discharged on the forty-sixth day in good condition.

Case V. This case is an example of relapse into a second decompensation while in the hospital, and after restoration following an initial period of decompensation.

This patient, W. L., male, age 52, had been discharged from the hospital restored from a period of cardiac decompensation only twenty-five days previous to his present admission. His stay in the hospital at that time was for a period of seventy days, and improvement was only slowly obtained. His maximum venous pressure during this period was 160 mms., and it was in the upper limits of normal (80 to 110 mms.) on discharge, although the edema and chronic passive congestion of lungs and liver, which was present on entrance, had subsided, and he was free from symptoms except on moderate physical exertion. Digitalis at a maintenance dose had been continued because of the presence of auricular fibrillation. Several days before his final admission he became very short of breath, and marked weakness developed. On this admission he was markedly orthopneic and cyanotic. Cheyne-Stokes respiration with apneic periods of 50 to 60 seconds was present. The pulse was totally irregular, with

an apical rate of 124, and a radial rate of 62. The apex beat of the heart was visible out beyond the midclavicular line. A systolic murmur, transmitted to the axilla, was present, and numerous râles were heard at both pulmonary bases. Systolic blood pressure was 100 mms. of mercury; venous pressure, 160 mms. of water. There was moderate edema of ankles and legs. The urine showed a high specific gravity, traces of albumin, and a few hyalin casts. The nonprotein nitrogen of the blood was normal. The blood Wassermann was negative. Phthalein urinary excretion was normal. The electrocardiogram showed auricular fibrillation and left ventricular preponderance, flattening of T_1, and negativity of T_2 and T_3.

The diagnosis was myocardial degeneration, cardiac decompensation, auricular fibrillation, chronic passive congestion of lungs and liver, and edema of extremities. Under rest, increased digitalis administration, fluid restriction, and venesection, rather rapid improvement occurred with diuresis, and marked reduction of edema and congestion. Venous pressure became approximately stabilized within normal limits. Following the seventy-first day, venous pressure began to rise and urine secretion to fall, and the clinical condition became rapidly worse (Fig. 7). Venous pressure rose to 170 mms., with a low urine output, marked increase of congestion and edema, and the patient again became acutely ill. Venous pressure reached a maximum of 230 mms. of

water on the eighty-ninth day, and on the ninety-first day, with a venous pressure of 220 mms., venesection was done with the removal of 250 cc. of blood. The venous pressure dropped sharply, urine output increased, and the general clinical condition improved. The improvement was not maintained, however, and the venous pressure rose somewhat irregularly with diminishing urine, and death oc-

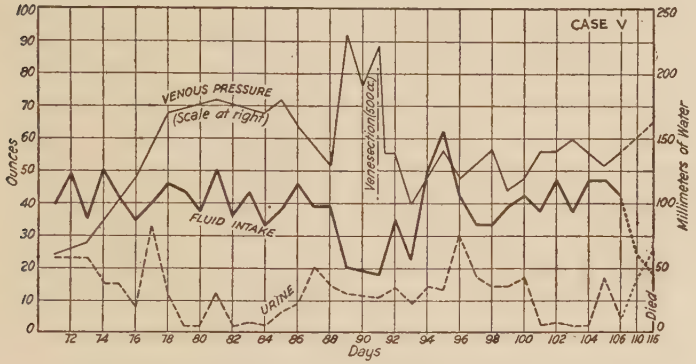


Fig. 7.—Case V. Similar to Fig. 3.

curred on the hundred and fifteenth day after admission. Autopsy revealed hypertrophy and dilatation of heart, fibrous myocarditis, pericardial effusion, adherent pericardium, aortitis with atheroma and calcification, acute broncho-pneumonia of right lower and middle lobes, and chronic passive congestion of lungs and liver.

Case VI. This case is included to illustrate the occurrence of cardiac decompensation in lobar pneu-

monia, and the associated cardiovascular findings. The clinical summary is quoted from a previous publication (2).

Mrs. K., aged 53, had a chill thirty-six hours previous to hospital admittance. There were signs of consolidation of the lower lobe of the left lung, and a systolic murmur at the apex of the heart. Blood pressure: systolic, 108; diastolic, 68 mms. of mercury; venous pressure, 80 mms. of water. The capillary circulation in the finger nail bed was normal. The pulse was 120. On the second morning in the hospital, the patient became much more toxic and dypsneic. Leukocytes, 16,000 per cubic millimeter. There was extension of consolidation to whole left lower lobe. The venous pressure was 90 mms. in the morning, rising to 130 mms. at 7:30 P.M. The capillaries showed contraction but no signs of stasis. Pulse, 120. The heart sounds were of good quality, but the pulmonic second sound was accentuated. Systolic pressure, 116 mms.; diastolic, 58 mms. At 11 P.M. large numbers of moist râles were heard over the chest. Involvement of the right lower lobe was present. The capillaries showed definite evidence of stasis. At six o'clock the next morning (third day in hospital) the patient was very cyanotic and ashen. The pulse was 116. There was no enlargement of the heart to percussion. The accentuation of the pulmonic second sound was less. Stasis in more than one-half the capillary loops was observed with slow streaming in others, and a general dilatation of

capillaries was present. The venous pressure was 170 mms. of water; systolic arterial pressure, 118 mms.; diastolic, 66 mms. of mercury. Two hours later there was marked increase noted in the extent of the pulmonary edema. Marked dyspnea was also present. Oxygen, strophanthin (intramuscular), and camphorated oil (intramuscular) were administered. The next morning (fourth day in hospital) the cyanosis was lessened. There was no important change in the pulmonary signs. Large and medium moist râles in great numbers were still present throughout both chests. There was no enlargement of the heart to percussion. The pulmonic second sound remained, however, somewhat accentuated. The venous pressure was 140 mms. Cyanosis was present, but less marked than yesterday. The pulse was of better quality, rate 112. Systolic pressure was 120 mms.; diastolic, 64 mms. In the afternoon of the same day the general condition was similar to that of the morning, but the venous pressure had fallen to 130 mms., and stasis in the capillaries was definitely less. On the next morning (fifth day in hospital), there was a great reduction in the number of large bubbling and other moist râles. There was no extension of consolidation. Cyanosis was much less marked. The dyspnea was much lessened. Pulse, 116; systolic pressure, 124 mms.; diastolic, 70 mms. Signs of stasis in capillaries were much less marked. The streaming of blood was faster and there was practically no beaded flow. The

venous pressure was 70 mms. In the afternoon of this day the venous pressure was 90 mms. of water; systolic arterial pressure, 128 mms.; diastolic pressure, 74 mms. of mercury. There were no large bubbling râles. The medium moist râles were lessened, and there was marked subjective improvement. The pulse rate was 120. On the next morning (sixth day in hospital) the condition was unchanged. Resolution was beginning in the lower lobes. Venous pressure, 90 mms.; systolic pressure, 128 mms.; diastolic pressure, 64 mms. Capillaries showed increased stasis. The venous pressure in the afternoon was 80 mms. with some reduction in the degree of capillary stasis. On the next morning (seventh day) signs of resolution were marked at both bases, but the lower portion of the right upper lobe was now involved. The pulse rate was 130; systolic arterial pressure, 132 mms.; diastolic, 68 mms. of mercury; venous pressure, 140 mms. of water. Signs of stasis in the capillaries were lessened. The pulmonic second sound was accentuated. At 10:30 P.M. venous pressure was 100 mms. of water; systolic arterial pressure, 116 mms.; diastolic pressure, 58 mms. of mercury. Only very slight evidence of stasis was present in the capillaries. Definite improvement was noted in the general condition on the following day (ninth day in hospital). The lungs were clearing throughout. The first heart sound was of excellent quality; the systolic murmur was louder. Pulse rate, 96; systolic arterial pressure, 110 mms.; dias-

tolic, 56 mms. of mercury; venous pressure, 40 mms. of water. There was very little, if any, sign of capillary stasis. Subsequent to the ninth day the gradual improvement continued to the twenty-sixth day, with no further signs of circulatory embarrassment. Twice daily observations of venous pressure gave readings varying between 30 and 70 mms. of water. A walled-off empyema sac in the left axilla was drained on the eleventh day, under ether anesthesia. No rise of venous pressure developed. Improvement following this operation was rapid, and the patient was discharged from the hospital on the fifty-fourth day. Frequent observations of the capillaries showed a normal flow throughout this period.

During the first five days the temperature ranged between 100.8° and 102° F. It then began a fall by lysis, and the first normal temperature (98.0° F.) occurred on the sixth day. An upward trend in the temperature then began, continuing until a second gradual decline on the eighth day. The leukocytes rose to a maximum of 29,000 per cubic millimeter on the sixth day, and then gradually declined. Fluids were taken in rather large quantities from the start, reaching a maximum of 3600 cc. on the third day. Urine output was much below fluid intake until the eighth day, and subsequently the two ran approximately parallel with the urine somewhat below fluid intake. During the period of pulmonary edema and circulatory embarrassment there was thus definite

fluid retention, with subsequent fluid loss as the condition of the circulation improved.

Teleroentgenograms for the determination of heart size were made on the sixth, seventh, ninth, twenty-first, and twenty-seventh days. Unfortunately, these do not include the first period of cardiac failure and high venous pressure, but they do include the second period of less marked decompensation extending through from the seventh to the ninth day inclusive. The five teleroentgenograms varied in area from 114 to 122 sq. cm.; the transverse diameter varied between 129 and 133 mms. These variations may be considered as falling within errors of technic, and as having no significance. Changes in the shape of the heart were also insignificant, the different teleroentgenograms being practically superimposable.

The stasis in the capillaries and rising venous pressure in this case, preceding the definite clinical evidence of cardiac incompetence, and the improvement in capillary flow and fall of venous pressure as compensation is reëstablished, are sufficiently evident to require no further comment.

CHAPTER VIII

Summary

Venous pressure in the normal individual is in part the pressure residue in the vascular system after the resistance in the peripheral vessels (arterioles, capillaries, and venules) is overcome. It is also in part the result of the fact that the end of the vascular system (the venæ cavæ) is not a free opening under zero pressure, but terminates in a closed organ, the heart, in which a positive pressure (except perhaps momentarily) always exists. The venous pressure at the heart determines the positive pressure to which the auricle and ventricle is exposed in diastole, and this under normal conditions determines the extent of the contraction of this organ and the amount of blood it expels. Within certain limits of venous pressure, limits defined by the ability of response on the part of the heart muscle, the pressure and ventricular output appear to bear a direct relation to one another. An increased venous pressure results in an increase in the amount of blood pumped by the heart into the vascular system. As a result of this, the heart tends to move the blood coming to it from the venous side into the arterial side without more than a transitory rise of venous pressure. The

normal heart muscle is capable of responding in this way to a wide range of venous pressure. Because of this, it is the most important factor in tending to stabilize venous pressure, and in preventing excessive variations. The erect posture and muscular exercise appear to affect venous pressure mainly by increasing the flow of blood through the peripheral vessels, and thus the amount returning to the heart. This increased flow with rise of venous pressure reacts upon the heart in the manner described above, and in a way which tends to limit the rise that would otherwise result from the increased peripheral flow. A constant and comparable venous pressure reading is, however, only obtained when these two factors are controlled, and determinations of venous pressure for clinical purposes should only be made under approximately basal conditions (recumbent posture and body rest).

The response of the normal heart to the venous load, as represented by the venous pressure, is adequate through wide limits. The pressure in the larger peripheral veins under basal conditions is ordinarily between 40 and 60 mms. of water, and rarely, if ever, exceeds 110 mms. The venous pressure in the heart itself is probably close to the same figure, because the tendency to a fall of pressure in the larger veins is counteracted by the negative intrathoracic pressure to which the venæ cavæ and heart are exposed. Under conditions of exercise or strain, these values may be increased several times

without evidences of cardiac embarrassment. Indeed, it seems probable that the venous pressure never rises in the normal individual to a point where it exceeds the capacity of the heart to respond to the increased load by increased work.

The condition in which the venous load has exceeded the capacity of the heart to respond by increased work, and in which the heart is failing to move the blood adequately from the venous side into the arteries, we call cardiac decompensation, or cardiac failure. There is evidence that this occurs only when the ability of the heart muscle to respond to increased loads is definitely narrowed as a result of disease of the muscle. Cardiac decompensation thus occurs only in the presence of heart muscle injury, either (and most frequently) as a result of degenerative processes superimposed on muscle hypertrophy, or as a result of toxemia (associated with pneumonia and more rarely other acute infections). Recent work indicates that the compensatory hypertrophy of organic heart disease has for its stimulus an injury process in the muscle fibers resulting from their mechanical stretching, and that there result changes which for a time increase the mechanical capabilities of the heart, but which ultimately lead to early degenerative changes, and a narrowing of the capacity of responding to large loads. The failure of the heart to move the usual quantity of blood results in further accumulation in the venous system, and a further rise of venous

pressure. The venous pressure, even under basal conditions, exceeds the normal, and may be twice or three times the upper normal limit. The heart is dilated under this excessive filling pressure, but, due to the fact that the pressure and dilatation have passed beyond the upper limit of physiological response on the part of the muscle, the contractions are weak and inefficient. This state continues until, as a result of rest, venesection, or other therapy, there is an improvement in the condition of the heart muscle, and a reduction of the venous load. In favorable cases, the venous load again comes within the range of response of the muscle; the heart by increased work reduces the abnormal venous congestion; and heart output and venous pressure return to within normal limits. This constitutes the return of compensation.

From theoretical considerations and practical experience, it is concluded that a sustained rise of general venous pressure above the upper normal limit under basal conditions, invariably accompanies cardiac decompensation, and furthermore that it is met with in no other clinical condition. Local venous obstruction may result in an abnormal rise of venous pressure in the region affected, but a generalized rise of venous pressure is characteristic of cardiac incompetence. This fact illustrates the usefulness, and at the same time the limitations, of venous pressure determination in clinical medicine.

Abnormal alterations of venous pressure occur in a relatively small clinical field, namely, in present or impending cardiac failure; but their determination and study are of paramount importance in this field.

The high venous pressure that accompanies cardiac incompetence is directly responsible for most of the signs and symptoms of this clinical state. Congestion of the lungs, liver, spleen, and other organs; diminished vital capacity of the lungs; and diminished urine secretion and water retention, are the result of venous and capillary engorgement and stasis. Careful and frequent determinations of venous pressure show that the changes in pressure in many cases precede the change in symptoms and signs, and may thus serve as an indicator of impending change. The progress of the case and the results of therapy are most accurately determined by a knowledge of the height, and especially the trend, of venous pressure. In Chapter III, methods for the clinical determination of venous pressure are considered, and it is shown that the indirect method of vein compression, especially, is accurate and easy to apply. An apparatus recently designed for clinical use is described in detail.

A detailed consideration of the relationship of venous pressure to the more important features of heart failure, such as the water balance and urine secretion, the dilatation of the heart in acute failure,

and the pressure and flow of blood in the capillaries, leads to the conclusion that these important results of cardiac incompetence are intimately associated with the abnormal venous pressure. Routine and frequent determinations of venous pressure furnish the best guide and control to the rational therapy of heart failure. Cardiac failure in lobar pneumonia shows the same relationship to venous pressure as the failure occurring in primary heart disease, and the importance of venous pressure determinations in this disease as a routine measure is emphasized.

Consideration of observations that have been made in clinical conditions other than cardiac failure, leads to the conclusion that has already been stated, but may be emphasized here, namely: that in the recumbent position, and at body rest, peripheral venous pressure is within normal limits, no matter what the clinical condition is, so long as the heart is competent.

An effort is made to emphasize throughout the fact that a knowledge of the venous pressure and a wider recognition of its relation and importance to cardiac incompetence, cannot but enlarge our conception of the more important of the clinical features of this condition. Through this, and by furnishing valuable information at the time in an individual case, and in a more quantitative manner than obtainable by other means, this knowledge must lead to a more intelligent and rational management of this important clinical state. Information of a

most helpful and important nature is obtainable, which up to the present has not received the attention and consideration that the welfare of the patient and the responsibility of the physician demand.

BIBLIOGRAPHY

1. v. Recklinghausen, H. Unblutige Blutdruckmessung. III. Messung des Blutdrucks in den kleinen Arterien, Venen und Kapillaren des Menschen und beim Thier. Arch. f. exper. Path. u. Pharm., 1906, lv, 463.
2. Eyster, J. A. E. and W. S. Middleton. Clinical studies on venous pressure. Arch. Int. Med., 1924, xxxiv, 228.
3. Bayliss, W. M. and E. H. Starling. Observations on venous pressure and their relation to capillary pressure. Journ. Physiol., 1894, xvi, 159.
4. Hooker, D. R. Functional activity of capillaries and venules. Physiol. Rev., 1921, i, 112.
5. Ebbecke, U. Gefässreaktion. Ergebn. d. Physiol., 1923, xxii, 401.
6. Krogh, A., G. A. Harrop and P. B. Rehberg. Studies on the physiology of capillaries. Journ. Physiol., 1922, lvi, 179.
7. Meek, W. J. and J. A. E. Eyster. Reactions to hemorrhage. Amer. Journ. Physiol., 1921, cvi, 1.
8. Meek, W. J. and J. A. E. Eyster. The effect of plethora and variations in venous pressure on diastolic heart size and the output of the heart. Amer. Journ. Physiol., 1922, cxi, 186.
9. Eyster, J. A. E. and W. S. Middleton. Cardiovascular reactions to hemorrhage and transfusion in man. Amer. Journ. Physiol., 1924, lxviii, 581.

10. Bainbridge, F. A. The influence of venous filling upon the rate of the heart. Journ. Physiol., 1915, xvi, 65.
11. De Graff, A. C. and J. Sands. Importance of reflexes from veins and auricles. Amer. Journ. Physiol., 1925, lxxiv, 415.
12. Wiggers, C. J. and L. N. Katz. The contour of the ventricular volume curve under different conditions. Amer. Journ. Physiol., 1922, lviii, 439.
13. Patterson, S. W. and E. H. Starling. On the mechanical factors which determine the output of the ventricles. Journ. Physiol., 1914, xlviii, 357.
14. Straub, H. Dynamik des Säugetierherzens. Deutsch. Arch. f. klin. Med., 1914, cxv, 531.
15. Eyster, J. A. E. Experimental and clinical studies in cardiac hypertrophy. Journal of the American Medical Association, 1928, 91, 1881.
16. Hooker, D. R. Observations on the venous blood pressure in man. Amer. Journ. Physiol., 1914, xxxv, 73.
17. Briscoe, G. Observations on the venous and capillary pressure with especial reference to the Reynaud phenomenon. Heart, 1919, vii, 35.
18. Keith, W. M., L. G. Rowntree, and J. T. Geraghty. A method for the determination of plasma and blood volume. Arch. Int. Med., 1915, xvi, 547.
19. Lundsgaard, C. Untersuchungen ueber das Minuten volumen des Herzens bei Menschen. II. Patienten mit Herzklappenfehlern. Deuts. arch. f. klin. Med., 1916, cxviii, 513.
19a. Henderson, Y. and H. W. Haggard. The circulation and its measurement. Amer. Journ. Physiol., 1925, lxxiii, 193.
20. Stewart, G. N. Studies on the circulation in man.

The blood flow in the hands and feet in normal and pathological cases. The Harvey Lectures, 1912-13, p. 86.

21. Krogh, A. The regulation of the blood supply to the right heart. Skand. Archiv. Physiol., 1912, xxiv, 227.
22. Barcroft, J. and J. G. Stephans. Observations upon the size of the spleen. Journ. Physiol., 1927, lxiv, 1.
23. Clark, A. A study of the diagnostic and prognostic significance of venous pressure observations in cardiac disease. Arch. Int. Med., 1915, xvi, 587.
24. Rhode, E. Ueber den Einfluss der mechanischen Bedingungen auf die Tätigkeit und den Sauerstoff Verbrauch des Warmblutherzens. Arch. f. exper. Path. u. Pharm., 1912, lxx.
25. Rhode, E. and S. Ogawa. Gaswechsel und Tätigkeit des Herzens unter dem Einfluss von Giften und Nervenreizung. Arch. f. exper. Path. u. Pharm., 1912, lxix, 200.
26. Hyde, I. The effect of distention of the ventricle on the flow of blood through the walls of the heart. Amer. Journ. Physiol., 1898, i, 215.
27. Morowitz, P. and A. Zahn. Untersuchungen ueber den coronar Kreislauf. Deutsch. Arch. f. klin. Med., 1914, cxvi, 364.
28. Nakogama, T. On the coronary circulation in the heart lung preparation. Journ. Physiol., 1922, lvi, 340.
29. Loevenhart, A. S., J. Y. Malone and H. G. Martin. Studies on stimulation of the respiration. The action of respiratory stimulants upon the respiration, when depressed by increased intracranial pressure with special reference to sodium cyanide. Journ. Pharm. Exper. Therap., 1922, xix, 13.

30. Frey, A. Ueber die Bedeutung der Venedruckmessung bei dietetishphysikalischer Behandlung des Kreislaufsstörungen. Deutsch. Arch. f. klin. Med., 1902, lxxiii, 511.
31. v. Basch, S. Ehrfahrungen ueber den Venendruck des Menschen. Arch. d. sci. biol., 1904-5, ii (suppl.), 117.
32. Hooker, D. R. and J. A. E. Eyster. An instrument for the determination of venous pressure in man. Johns Hopkins Hosp. Bull., 1908, xix, 274.
33. Hooker, D. R. The influence of age upon the venous blood pressure in man. Amer. Journ. Physiol., 1916, xl, 43.
34. Gaertner, G. Die Messung des Drucks in rechten Vorhof. Münch. Med. Wochenschr., 1903, l, 2038.
35. White, H. L. Observations on venous pressure and skin blanching pressure. Amer. Journ. Physiol., 1924, lxix, 10.
36. Frank, L. and M. Reh. Eine graphische methode zur unblutigen Bestimmendes Venendruck in Menschen. Zeitschr. f. exper. Path. u. Therap., 1912, x, 24.
37. Moritz, and v. Tabora. Ueber ein Methode, beim Menschen der Druck in oberflächlichen Venen exactzer Bestimmen. Deutsch. Arch. f. klin. Med., 1910, xcviii, 475.
38. Schott, E. Die Erhöhung des Drucks in venösen System bei Anstrengung, als Mass fur die Funktionstuchtigkeit des Menschlichen Herzens. Deutsch. Arch. f. klin. Med., 1912, cviii, 537.
39. Claude, H., R. Porox and J. Rouillard. Recherche de manometrie clinique avec application particulurment a l'étude de la pression du liquide ceppalo-rachiden. Revue de Med., 1914, 393.

40. Fuchs, L. Ueber die Messung des Venendrucks und ihre klinische Bedeutung. Deutsch. Arch. f. klin. Med., 1921, cxxxv, 68.
41. La Conte, M. and J. Yacoel. La tension veineuse a l'état normal et pathologique. Journ. de Med. et de la Chir. Pratique, 1922, xciii, 171.
42. Villaret, M., Fr. Saint Girons and Grellity-Bosviel. Contribution a l'étude de la tension veineuse périphérique a l'état normal et pathologique. Le Journ. Med. Français, 1921, x, 359.
43. Gazzotti, J. Ricerche sulla pression venosa perifica mesurata nell'uomo. Mallattio del cuore e die Vas., 1923, vii, 36.
44. Castellotti, Fr. Contributo allo studio clinica della pression venosa. Mallattio del cuore e die Vas., 1927, vii, 69, 277, 317.
45. Bedford, D. E. and S. Wright. Observations on venous pressure in normal individuals. Lancet, 1924, ii, 106.
46. Runge, H. Ueber den Venendrucks in Schwangerschaft, Geburt, und Wochenbett. Arch. f. Gynak., 1924, xxii, 142.
47. Blumgart, H. L. and S. Weiss. Relation between velocity of blood flow, venous pressure and vital capacity of lungs in fifty patients with cardio vascular disease compared with similar measurements in fifty-eight normal persons. Journ. Clin. Invest., 1928, v, 343 and 379.
48. Barach, J. H. and W. L. Marks. Effect of change in posture, without active muscular exertion on the arterial and venous pressures. , Arch. Int. Med., 1913, xi, 485.
48a. Henderson, Y., A. L. Prince and H. W. Haggard. The circulation in man in the head-down position and a method for measuring the venous return

to the heart. Journ. Pharm. Exper. Therap., 1918, xi, 189.

49. White, H. L. and R. M. Moore. Circulatory response to static and dynamic exercises. Amer. Journ. Physiol., 1925, lxxiii, 636.
50. Hooker, D. R. The effect of exercise on the venous pressure. Amer. Journ. Physiol., 1911, xxviii, 235.
51. McCrea, F. D., J. A. E. Eyster and W. J. Meek. The effect of exercise upon diastolic heart size. Amer. Journ. Physiol., 1928, lxxxiii, 678.
52. Burton-Opitz, R. The venous supply of the heart. Amer. Journ. Physiol., 1921, lviii, 226.
53. Kroetz, C. Die Koeffizienten des klinische messbaren Venendrucks. Deutsch. Arch. f. klin. Med., 1922, cxxxix, 325.
54. Waud, R. A. The effect of respiration on the venous pulse as studied by the electropolygraph. Amer. Journ. Physiol., 1924, lxxvi, 112.
55. Henderson, Y., A. L. Prince and H. W. Haggard. Influence of forced breathing on the circulation. Journ. Pharm. Exper. Therap., 1918, ii, 103.
56. Burger. Ueber die Klinische Bedeutung des Valsavaschen Versuch. Münch. Med. Wochenschr., 1921, lxviii, 1066, Situngsbericht.
57. Dawson, P. M. and P. C. Hodges. Cardiovascular reaction in the Valsalva experiment and in lifting with a note on parturition. Amer. Journ. Physiol., 1921, i, 481.
58. Ratky, H. and O. Klein. Studien ueber Venendruck und Kreislaufssufficienzprüfung nach E. Weiss nebst einem Beitag ueber die Ursachen der Steigerung des Venendrucks bei Hypertonikern. Med. Klinik, 1923, xix, 1574.
59. Eyster, J. A. E. and E. C. Swarthout. Experimen-

tal determination of the influence of abnormal cardiac rhythms on the mechanical efficiency of the heart. Arch. Int. Med., 1920, xxv, 317.

60. Eyster, J. A. E., F. J. Hodges and W. J. Meek. Cardiac changes subsequent to experimental aortic lesions. Arch. Int. Med., 1927, xxxix, 536.
61. Eyster, J. A. E. Cardiac dilatation and hypertrophy. Trans. Assoc. Amer. Physicians, 1927, xlii, 15.
62. Romberg, E. and A. Hesenfeld. Ueber die Reservekraft des hypertrophischm Herzmuskel. Arch. Exper. Path. und Pharmak., 1897, xxxix, 332.
63. Wolfer, P. Experimentelle Studien zur Reservekraft des hypertrophischen Herzens. Archiv. f. exper. Path. u. Pharmak., 1912, lxviii, 435.
64. Addis, T. and C. K. Watanaba. The volume of urine in young healthy adults on a constant diet. Journ. Biol. Chem., 1916, xxvii, 267.
65. Weir, J. F., E. E. Larson and L. G. Rowntree. Studies in diabetes insipidus, water balance and water intoxication. Arch. Int. Med., 1922, xxix, 306.
66. White, H. L. On glomerular filtration. Amer. Journ. Physiol., 1924, lxviii, 523.
67. Landis, E. M. The capillary pressure in frog mesentery as determined by micro-injection methods. Amer. Journ. Physiol., 1926, lxxv, 548.
68. Gasser, H. S. and J. Erlanger. Studies in secondary traumatic shock. V. Restoration of the plasma volume and of the alkali reserve. Amer. Journ. Physiol., 1919, l, 104.
69. Nevermann, H. Capillardruck messungen. Klin. Woch., 1924, xxxiii, 1433.
70. Friedlander, S. O. and C. H. Lambert. Clinical

observations on the capillary circulation. Arch. Int. Med., 1922, xxix, 12.

71. Lundsgaard, C. Studies on oxygen in the venous blood. I. Technique and results on normal individuals. Jour. Biol. Chem., 1918, xxxiii, 133.
72. Lundsgaard, C. Studies on the oxygen in the venous blood. IV. Determinations on five patients with incompensated circulatory disturbances. Journ. Exper. Med., 1918, xxvii, 219.
73. Hürter. Untersuchungen an arteriellen menschlichen Blute. Deut. Archiv. klin. Med., 1912, cviii, 1.
74. Landis, E. M. Microinjection studies. Effect of lack of oxygen on permeability of capillary wall to fluid and to plasma proteins. Amer. Journ. Physiol., 1928, lxxxiii, 528.
75. Richards, A. N. and O. H. Plant. Urine formation in the perfused kidney. The influence of alterations in renal blood pressure on the amount and composition of urine. Amer. Journ. Physiol., 1922, lix, 144.
76. Van Valzah, R., E. D. McKinley and W. S. Middleton. To appear in the Amer. Journ. Med. Sci.
77. Wilson, J. A. and W. J. Meek. The effect of the pericardium on cardiac distention as determined by the x-ray. Amer. Journ. Physiol., 1927, lxxxii, 34.
78. Meek, W. J. The effect of changes in pulse rate on diastolic heart size. Amer. Journ. Physiol., 1924, lxx, 385.
79. Bardeen, C. R. Determination of the size of the heart by means of the x-ray. Amer. Journ. Anat., 1918, xxiii, 423.
80. Danzer, C. S. and D. R. Hooker. Determination

of the capillary blood pressure in man with the microcapillary tonometer. Amer. Journ. Physiol., 1920, lii, 136.

81. Plumier, L. Variations de la pression veineuse. Arch. Internat. de Physiol., 1909, viii, 1.
82. Weiss, E. Ueber Beobachtung der Haut Kapillaren und ihre klinische Bedeutung. Münch. Med. Wochenschr., 1917, lxiv, 609.
83. Liebesny, P. Untersuchungen ueber die Capillar druckmessung. Arch. f. d. gesamte Physiol., 1923, cxlviii, 215.
84. Rajka, E. and E. Wassely. Ueber das Verhalten des Capillerdrucks unter Physiologischen und Pathologischen Verhaltnissen, gemesen mit dem Töräk-Rajka-Wesselyschen Capillartonometer. Zeit. f. d. gesamte experimentelle Medizin, 1927, lvii, 171.
85. Danzer, C. S. Retrograde capillary pulsations in heart failure. Amer. Journ. Med. Sci., 1926, ccxxi, 654.
86. v. Kries. Berl. d. Sachs. ges. d. Wissensch. Math. Physiol., Kl., 1875, xxvii, 179.
87. Goldschmidt, S. and A. B. Light. A cyanosis unrelated to oxygen saturation, produced by increased peripheral venous pressure. Amer. Journ. Physiol., 1925, lxxiii, 173.
88. Lindhard, J. Effect of posture on the output of the heart. Skand. Arch. f. Physiol., 1913, xxx, 395.
89. Field, H. and A. V. Bock. Orthopnea and the effect of posture on blood flow. Journ. Clin. Investi., 1925-26, ii, 67.
90. Turner, A. H. The circulatory minute volume of healthy young women in reclining, sitting, and standing positions. Amer. Journ. Physiol., 1927, lxxx, 601.

91. Hamilton, W. F., J. W. Moore and J. M. Kinsman. Delay of blood in passing through the lungs as an obstacle to the determination of CO_2 tension of the mixed venous blood. Amer. Journ. Physiol., 1927, lxxxii, 656.

92. Moore, J. W., W. F. Hamilton and J. M. Kinsman. Ethyl iodide method for determining the circulation. Journ. Amer. Med. Assoc., 1926, lxxxvii, 817.

93. Hill, L. The influence of the force of gravity on the circulation of the blood. Journ. Physiol., 1895, xviii, 15.

94. Mobitz, W. Die Ermittlung des Herzschlagvolumens des Menschen durch Einatmung von Athyljodid. Klin. Wochenschr., 1926, v, 985.

95. Keith, N. L., L. G. Rowntree and J. T. Geraghty. A method for the determination of plasma and blood volume. Arch. Int. Med., 1915, xvi, 547.

96. Villaret, M., Fr. St. Girons and Grellity-Bosviel. La tension veineuse périphérique et ses modificationes pathologique. La Presse Medical, xxviii, 1.

96a. Eyster, J. A. E. Venous pressure and its clinical applications. Physiol. Rev., 1926, vi, 281.

97. Eyster, J. A. E. and W. S. Middleton. Venous pressure as a guide to venesection in congestive heart failure. Amer. Journ. Med. Sci., 1927, clxxiv, 486.

98. Gordon, B. The value of venesection in the treatment of the decompensated heart. Amer. Journ. Med. Sci., 1925, clxx, 671.

99. Capps, J. A. and S. A. Mathews. Venous blood pressure as influenced by the drugs employed in cardiovascular therapy. Journ. Amer. Med. Assoc., 1913, lxi, 338.

100. Cohn, A. E. and H. J. Stewart. Evidence that digitalis influences contraction of the heart in man. Journ. Clin. Investi., 1924, i, 97.
101. Burwell, C. S., D. Neighbors and E. M. Regan. The effect of digitalis upon the output of the heart in normal men. Journ. Clin. Investi., 1927, v, 125.
102. Cohn, A. E. and H. J. Stewart. The relation between cardiac size and cardiac output per minute following the administration of digitalis. Journ. Clin. Investi., 1928, vi, 53 and 79.
103. Stewart, H. J. and Gilchrist, Studies on the effect of cardiac irregularity on the circulation. II. The estimation of cardiac output in dogs subject to artificial auricular fibrillation. Journ. Clin. Investi., 1928, v, 335.
104. Withering, H. An account of foxglove and some of its medicinal uses with practical remarks on dropsy and other diseases. 1785, Birmingham.
105. Mackenzie, J. New methods of studying affections of the heart. IV. Action of digitalis on the human heart in cases where the inception of rhythm of the heart is due to the ventricles. Brit. Med. Journ., 1905, i, 759.
106. Cushny, A. R. The therapeutics of digitalis and its allies. Amer. Journ. Med. Sci., 1911, cxli, 469.
107. Cohn, A. E. Clinical and electrocardiagraphic studies in the action of digitalis. Journ. Amer. Med. Assoc., 1915, lxv, 1527.
108. Christian, H. A. Digitalis therapy: Satisfactory effects in cardiac cases with regular pulse rate. Amer. Journ. Med. Sci., 1919, clvii, 593.
109. Marvin, H. M. The value of the xanthin diuretics in congestive heart failure. Journ. Amer. Med. Assoc., 1926, lxxxvii, 2043.

110. Levy, R. L. The size of the heart in pneumonia. Arch. Int. Med., 1923, xxxii, 359.
111. Henderson, Y. The volume curve of the mammalian heart, and the significance of this curve in respect to the mechanism of the filling of the ventricles. Amer. Journ. Physiol., 1906, xvi, 325.
112. Ayerza, L. La tension venosa perifica (P.V.) Sus Modificaciones patalogicos y valer pronostico. Samana Medica, 1924, xxiv, 1921.
113. Cuneo, B. Sept cas d'aneurismes artério-veineuse des vaissaux femoraux. Bull. et Memoirs de la Soc. de Chir., 1917, xxiv, 1921.
114. Fahr, G. and E. Ronzone. Circulatory compensation for difficult oxygen carrying capacity of the blood in severe anemias. Arch. Int. Med., 1922, xxix, 331.
115. Helouin, M. La tension intraveineuse chez les hypotendus et hypertendus artériels. La Presse Medicale, 1925, xxxiii, 1378.
116. Tatterova, R. and J. Serf. Časopis Líkăru Českýeh. Prague, 1925, lxiv, 1364.
117. Villaret, M. and Martiny, M. Pression veineuse et tuberculose pulmonaire. La Presse Medical, 1925, xxxiii, 1569.
118. Markovitch, V. Pression veineuse au cours de l'asystole. These de Paris, 1923.
119. Frank, L. Ueber den Ausgerich des arterillen und venosen Drucks in aus den Blutbahn ausgeschalteten Theilen des Gefassystems. Zeitschr. f. exper. Path. u. Therap., 1913, xiii, 37.
120. Villaret, M., Fr. Saint Girons and L. Salasc. Tension veineuse et varices. Compte Rendu et Memories de la Societé de Biologie, 1925, xciii, 230.
121. Middleton, W. S. A case of aneurism of the aorta

with unusual pressure signs. Wis. Med. Journ., 1922, xxi, 37.

122. Villaret, M., Fr. Saint Girons and Grellity-Bosviel. Contribution a l'étude de la tension veineuse périphérique. La syndrome d'hypertension veineuse et d'acrocyanose avec insufficsance ovarienne. Bull. et Memoires de la Soc. de Hôpitaux de Paris, 1921, xlv, 1013.

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on or before the date last stamped below.

SEP -1 1930
MAY 23 1931
JUN 10 1931
AUG 26 1931
OCT 30 1931
DEC - 1 1931
DEC 25 1931
APR -1 1932
MAY 12 1932
DEC 23 1932
Mar 8
MAY 27 1933
JUL 20 1933
MAY - 9 1935
AUG 25 1936
JAN - 3 1944
AUG 26 1944
SEP - 8 1947

L669 Eyster, J.A.E. 71167
E97 The clinical aspects of
1929 venous pressure.

NAME	DATE DUE
Gelpiani	AUG 25 1936
Hultgren	JAN 3 1944
Bob Livingston	AUG 26 1944
V. Gilbert	SEP -8 1947
ren	OCT 1947
ren	NOV 15 1947
ren	DEC 18 1947